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"Scire est nescire, nisi id me Scire alius sciret."

WINTER, 1934.

THE TWENTY-THIRD LONG FOX MEMORIAL LECTURE:

DELIVERED IN THE UNIVERSITY OF BRISTOL ON TUESDAY, JULY 3RD, 1934.

THE VICE-CHANCELLOR (Dr. T. LOVEDAY, M.A., LL.D.) *in the Chair.*

BY

F. J. POYNTON, M.D., F.R.C.P.,

Consulting Physician to University College Hospital and to the Hospital for Sick Children, Great Ormond Street.

ON

SOME ASPECTS OF HEART DISEASE IN CHILDHOOD.

IT gives me unusual pleasure to have been honoured by the invitation to give the Long Fox Lecture, and to follow in the paths of those distinguished men who have already held this post.

Once more I see myself a lad of seventeen walking from St. Philip's Station across Bristol Bridge and the Drawbridge, as it was in those days, then upward

past the Merchant Tailors' School to the University College. There was no University with its commanding tower, but there were great teachers in that college —Professor Lloyd-Morgan, Sir William Ramsay, Dr. Young and Professor Leipner—who taught me the love of scientific knowledge and the methods of scientific inquiry. After many years I returned here as an External Examiner in Medicine, and to-day I am acting as your Lecturer.

Needless to say, I have thought much as to the best use I could make of such an opportunity, and finally decided to fall back on my personal experience in London of organic heart disease in childhood.

You must not think that I have only concerned myself with heart disease in childhood, for I have been in charge of adults also at University College Hospital for over thirty years. One of my chief concerns with children has been to investigate the origin and possible prevention of early organic heart disease, and my experience with adults has been a continual incentive to pursue that study.

It is not my intention to weary you with the pathological investigations I made of rheumatic carditis and malignant endocarditis, of diphtheria and septic infections, or with the investigations into the cause of acute rheumatism made with my late friend and great research worker Dr. Alexander Paine. Some of these were made in the last century, and have been elaborated or improved upon or passed over.

I should not, however, be doing due honour to the University College from which I received my early training if I did not claim that these investigations anticipated the Aschoff's nodules, demonstrated the essential damage done to the myocardium by rheumatism, and contributed the first experimental

proof in this country of the production of carditis from the active lesions of acute rheumatism.[1,2,3] It is, I think, remarkable that a Bristol physiologist, Dr. Kent, in 1892 demonstrated clearly and decisively what is now called the bundle of His, and that the further work of the late Dr. Carey Coombs, Dr. C. E. K. Herapath, Professor G. Hadfield and Dr. Bruce Perry on the rheumatic heart has again carried on the advance of our knowledge of this subject. Moreover, Bristol has been the pioneer in the provinces by establishing at Winford a school hospital.

I have no doubt that to the teaching at the school of Bristol will in the future be given the credit for much of the pioneer work done in this country on the myocardium, and no one will deny that the myocardium is the main factor in determining the course of most cases of organic heart disease.

RHEUMATIC HEART DISEASE.

We do not know the nature of the poisons of rheumatism, but clinical experience has taught us that these must differ much in virulence in different children. Thus strong children of about ten years of age may recover so wonderfully from an acute carditis that years later no one could believe that there had been a severe pericarditis. On the other hand, rheumatism may wreck the myocardium, and the autopsy demonstrates that the valvular damage could not have greatly injured the function of the heart, so slight was it in its degree.

It is not sufficiently realized that acute so-called "simple" rheumatic endocarditis is not fatal, so that when death occurs from a general carditis there has been time for the infective micrococci of rheumatism to be destroyed in the vegetations on the valves, or if

not destroyed to require the utmost care and patience in their discovery, and the same is true of experimental heart disease. The exception to this is malignant rheumatic endocarditis, which most observers do not recognize, claiming that this form of endocarditis is always the result of a secondary infection, a question to which I will revert later. It is, I also think, only those who have studied the infection in animals and traced it through every stage who can realize how effectually the tissues, cells and leucocytes combine to destroy the strepto-diplococci in the tissues.

However, my intention is not to dwell upon pathology, but upon the more clinical aspect of organic heart disease in childhood as I have seen it in London.

The rheumatic type varies much in frequency and virulence, not only in individual cases but in different years. Last year, in the autumn, after the long, dry summer, the cases were severe, and I never saw subcutaneous nodules so frequently, nor that sub-periosteal inflammation over bony prominences which leads to the "growing out," as I term it, of the bones of the wrists, of the elbows and ankles. In one case a bony node was detected on the shaft of the left femur, such as I have seen produced by experiment from an intravenous inoculation of a rabbit with the strepto-diplococcus. It is many years since Sir Arthur Newsholme insisted that rheumatism was essentially an urban disease, rife when the sub-surface level of water was low. During last autumn there were also extensive outbreaks of tonsillitis in London, and we may be sure that when these outbreaks occur there will be much acute rheumatism among London children as the bright, warm weather swiftly turns to grey cold.

Apyrexial Carditis.

The most deadly form of rheumatic heart disease is the apyrexial type, with a stealthy carditis, nodules and rapid anæmia. I know no remedy for it—salicylates, tolysin, rest and splendid nursing all fail. Fortunately it is not very frequent. Not only may it be deadly, but it is easily underrated when it does occur, and the inexperienced, missing the cardiac dilatation, do not realize the importance of the rapid pulse, and may easily enough overlook a warning subcutaneous nodule. This type chiefly affects the very young, and I should venture to traverse the frequent statement that acute rheumatic heart disease is rare under five years. I have repeatedly seen it occur under that age, and taking a hundred consecutive cases of varying severity in my ward I found 10 per cent. began before the age of five years. Doctors should not be led astray by the belief that this early rheumatism is most exceptional, and must not forget that the mortality at that age is in proportion greater than at any period.

Hyperpyrexia.

This dreadful apyrexial type becomes the more arresting and remarkable when contrasted with the case of a boy of fifteen years who came under my care last year wildly delirious and incontinent, with a temperature of over 106° F., carditis and arthritis. He, in spite of general pericarditis, aortic and mitral disease, has recovered so marvellously that it is with difficulty now that any murmur at all can be detected, and he is back at work, cheerful and strong.

Hyperpyrexia in rheumatism is not a thing of the past, for in addition to this case I have recently seen two cases of chorea with rheumatic arthritis and a

temperature of nearly 107° F. These reacted well to cold sponging.

It cannot be too strongly insisted that to look on lowering of the temperature as any proof of the specific efficacy of salicylates in the acute rheumatism of childhood is fallacious, seeing that the most virulent cases are apyrexial ; but it is difficult indeed to convince students and even doctors that the intensive—I stress the word "intensive"—treatment of acute carditis in early childhood with salicylates is not only unsuccessful but may be fatal.

Venous Thrombosis.

There is one event in the rheumatic heart disease of childhood that has always interested me, and upon which I published an early investigation in 1898.[4] My interest has been lately refreshed by a Bristol physician, Dr. Bruce Perry, one of my former house-physicians at Great Ormond Street, who independently in this city confirmed these observations.[5] This event is the occurrence of thrombosis of large veins. I published originally three cases, but I have seen ten examples, and have not a shadow of doubt as to its reality, though few other observers in this country have recorded its occurrence. The axillary and innominate veins have in my experience been the most frequently affected, but one of my first cases, which proved fatal, showed thrombosis of the superior vena cava and all its main tributaries, death occurring from a cardiac embolism.

Those who have not seen this condition may object that it is a mechanical result, brought about by a severe pericarditis with adhesions around the large veins entering the heart, aided by the failing circulation. A. F. Voelcker made this explanation unlikely when he

described a case of chorea (and chorea in this country as a rule is undoubtedly an active rheumatism) in which, although there was no cardiac disease, there were thromboses in the femoral and saphena veins.[6] Again, in 1927, in a paper with the rather fantastic title "Inspiring Cases of Rheumatism,"[7] I described a case of severe chorea with carditis and venous thrombosis of the inferior vena cava with recovery. There was later the development of that striking collateral circulation such as we sometimes see after the same event in typhoid fever.

Clearly, then, we can put aside once and for all an explanation based on a mechanical interference associated with pericardial adhesions. This was to me evident from my first observations, though these were unfortunately made before I had commenced investigating the streptococcal cause of acute rheumatism. The evidence lay in the fact that the veins in which the thrombosis occurred showed acute inflammation and changes comparable to those so carefully described by Dr. Bruce Perry. These resemble those occasionally seen in the rheumatic arteritis of childhood, of which I have a striking example, made from a section of the aorta of a child who died of virulent rheumatism at the age of three years. Whether or not the phlebitis is due to some remarkable change in the blood or is due to a local infection may be disputed, but I incline to the latter view. The diagnosis is easy, and the more easy because everyone will admit that cardiac œdema is not a striking feature of acute carditis in childhood. Indeed, one of the remarkable features of the œdema in this acute carditis is a slight puffiness of the face, which suggests a renal rather than a cardiac œdema, although there is no evidence of renal disease that one recognizes. If, then, we come to the bedside of one

of these cases and find an unmistakable unilateral swelling of an arm and hand the diagnosis is instantly clear, and if the external and internal jugular veins are affected the tender hard cords of the veins in which the thrombosis has occurred will be palpable.

Similarly, if we find both lower extremities rapidly becoming much swollen with œdema we know that unless it is the final stage of congestive failure due to chronic rheumatic valvular disease, in an acute carditis it must be due to a venous thrombosis.

These cases are not all fatal. On the contrary, five I have watched recovered from the thrombosis, though cardiac lesions may be left of varying degrees of severity, and in my experience they are generally severe.

In passing I would mention that in acutely virulent cases of rheumatism you may not find Aschoff's nodules, just as in virulent acute tuberculosis you may not find any giant cell formation.

The earliest focal lesion in virulent rheumatism affecting the heart I consider to be an area of necrosis in the connective tissue, and around this develop the Aschoff nodules, which I interpret as the first stage in the healing of the focal lesion. This I would compare with the subcutaneous nodule, which in the early stages shows on section a central small core of necrosis, a middle area of cellular infiltration, and an outer zone of swollen connective tissue with distended blood capillaries. The central core of necrosis is in the cardiac lesion minute, and as in the subcutaneous nodule the amount of scar formation is very small, provided the rheumatic process is not prolonged and recurrent, when there may result a considerable fibrosis. I distinguish between these focal connective

tissue lesions and the general damage to the myocardium by the rheumatic poison.

Heart Block.

Under this heading I will content myself with citing one case which illustrates the intense interest that surrounds early rheumatic heart disease. There is no point about it that has not been recorded by others, but its value is in the completeness of the history.

I saw this patient when a boy aged 13 years, in 1926. He had a sore throat and had fainted several times, had vomited and complained of pain in his chest. He was a nervous, clever, athletic boy who had usually excellent health. When I saw him in bed his throat was inflamed, his pulse 36 to the minute, but the jugular pulsations were 72, and I diagnosed heart block with probable Stokes-Adams symptoms. He was transferred to hospital for investigation, but the heart block disappeared and a slight mitral systolic murmur became audible. This was followed by a formidable attack of acute rheumatism with multiple arthritis, chorea and aortic and mitral valvulitis. He was completely dumb with the chorea and took months to recover from his illness, leaving hospital with a mitral lesion and aortic regurgitation. He then apparently remained well, subsequently passed his B.Sc. of London, and obtained a scientific post of some promise. Before, however, he actually commenced this work he complained of breathlessness, became anæmic, and had arthritic and muscular pains for which he came to see me at the hospital. He had grown well in the interval, was most intelligent, and had never been laid up since his severe illness. The anæmia was obvious, the valvular lesions with the aortic predominant were present, the temperature was raised, and the heart excited in action. On the tips of his fingers were Osler's spots, which he had himself noticed. The diagnosis of acute malignant endocarditis was only too obvious, and shortly afterwards a large cerebral embolism occurred, followed by death.

Here we have this sequence: the well-known but comparatively rare commencement of acute rheumatism

with heart block following a sore throat, a fulminating acute attack of the disease, a period of quiescence and then, without any obvious cause, the final tragedy of fatal acute malignant endocarditis. This is the only case I have traced from an early passing heart block to death, although we know that death may follow from a general carditis in such cases, but I have traced many examples of rheumatic aortic and mitral disease to death from malignant endocarditis, to which I now turn.

Malignant Endocarditis.

There are few more vitally important and interesting problems concerned with rheumatism than that of malignant endocarditis, and there is not one of us who has not felt the utter hopelessness and futility of our efforts to arrest its progress.

We know there are many causes of malignant endocarditis, and we know, too, that it is comparatively infrequent in childhood; we know also that in the rheumatic it is generally looked upon as a secondary infection (usually by streptococci) of a damaged valve or valves. The mention of streptococci at once makes us think carefully, particularly if we admit that a streptococcus is a cause of acute rheumatism.

Again, we have heard and read much of the streptococcus viridans and hæmolyticus as studied in the bacteriologist's laboratory, but for a few moments I would venture to direct attention to a clinical side of acute rheumatism.

Many years ago, in 1899, I published a case of very extensive purpura during an attack of acute rheumatism,[8] which at autopsy showed the lesions that are accepted as those of this disease and from which during life Dr. A. Paine isolated a streptococcus. From time to time since I have seen considerable

purpura in acute rheumatism, and have also seen rheumatic nodules surrounded by zones of purpura. The most frequent cause of a hæmorrhagic pericardial exudation in childhood is acute rheumatism. Epistaxis is a well-known symptom, and I personally believe a hæmorrhagic nephritis may occasionally be rheumatic; and Dr. Paine experimentally produced a hæmorrhagic nephritis with the strepto-diplococcus.

It is clear to me, then, from clinical study that if rheumatism is streptococcal in origin the streptococcus must be certainly at times hæmolytic. On the other hand, not all cases of rheumatism in childhood show hæmorrhagic symptoms, yet develop the classical lesions, and thus it appears to me also that there is no cardinal distinction to be made in clinical medicine between a streptococcus which is hæmolytic and non-hæmolytic if this be expressed in terms of human disease. I would interpret this as meaning that there are factors, not yet understood, dependent not only on the streptococcus, but the individual attacked, which govern the phenomenon of hæmolysis. In other words, I am not yet convinced that we can judge the exact rôle in human disease taken by the streptococcus when it is once isolated from the human tissues and studied *in vitro*.

The point of this digression is this: that it is possible to me that a streptococcus lurking in the system may change its characters when those tissues are altered by general ill-health or bad surroundings, or even by long sojourn in human tissues, and that the rigid view of a secondary infection to account for malignant endocarditis is not to be accepted.

If it is accepted, why is malignant endocarditis uncommon in childhood, the age at which acute rheumatism is rife? Have not children repeated

occasions for secondary infections? Do they not, many of them, live in stuffy rooms, and are not many under-nourished? Why, then, is malignant endocarditis infrequent in childhood, seeing that many rheumatic children have sores or otitis or mastoiditis or nasal suppurations? Rheumatic children in my opinion are relatively more often exposed to secondary infection than the adult, and for me this rigid view of secondary infection palpably fails to explain the clinical fact of the comparative rarity of malignant endocarditis in the child.

Turning to microscopy, what do we sometimes find when we carefully and persistently examine many sections of a rheumatic endocarditis? We find in acute cases the streptococci in what I call the "fighting area," that is to say, where the area of necrosis in a vegetation meets the live part of the valve in which there is active leucocytosis and tissue reaction. These are scanty, they may be scanty even in experimental endocarditis (which is a crude and gross infection), for the organism is rapidly destroyed by the tissues, or I would rather say destroyed so far as our staining methods can show. Again, we may find in chronic cases in the human being small areas of necrosis in a rheumatic valve shut off more or less imperfectly by fibrous tissue.

Two questions arise. First, are we certain when a rheumatic endocarditis has completely healed and, if so, how are we certain? Secondly, are we sure that the streptococci, though not visible in the necrotic area of a rheumatic valve, are certainly destroyed or only that they lose their power to retain stains? In fact, are "destruction" and "loss of staining power" interchangeable terms? Again, the streptococci may be lurking in the immediate neighbourhood, not in

the necrotic tissue but in the living valve tissue itself.

In order to emphasize the importance of these questions, I must turn for a moment from actual childhood to a young medical student who died of malignant endocarditis. He had, when at hospital, an acute rheumatic attack, which left him with severe aortic regurgitation and a mitral lesion. Later, after leaving hospital, he would come to see me at intervals whenever he had warnings of fresh rheumatism. He was clever and became a most promising doctor. Every time he came to see me he said: "I believe I have malignant endocarditis," and every time I laughed away his fears, which were also mine. He later settled down to work for about two years free of definite rheumatism, but with severe valvular lesions, a very large heart and occasional arthritic pains. At last, without any obvious reason—and how often, I ask, is the reason obvious?—he developed more rheumatic pains, and became very tired and breathless, and there was some fever. Streptococci were found in the blood, and the fear had now become a certainty. He had malignant endocarditis. While under observation during that final illness he developed an acute arthritis affecting his knees, wrists and hands. This gave the poor fellow great hope, for we had not let him know the truth we feared, and in that development he saw a return of his old enemy, acute rheumatism, which I believe it was. This arthritis with effusion subsided completely under treatment, but he died of the malignant endocarditis.

I feel morally certain that through the five years after his first attack of acute rheumatism those cardiac valves had never really healed, and I do not believe there was a secondary, non-rheumatic infection.

In children I have seen rheumatic pericarditis subside and multiple arthritis vanish, and yet death occur in the same illness from malignant endocarditis. Are we to assume two simultaneous infections with two different streptococci, one only attacking the valves of the heart, the other the pericardium and joints?

I have cut rheumatic nodules and demonstrated streptococci in diplococcal form in the "fighting zone."

I have cut a large subcutaneous nodule over the elbow in malignant endocarditis, and demonstrated streptococci in diplococcal form in the "fighting area," and in a blood capillary just external to that area.

I have shown at the Medical Society of London the heart[9] of a child who had died from severe carditis with multiple arthritis. The vegetations on the aortic cusps were classically rheumatic, and they were the same on the mitral valve except for one, and this was a large fungating vegetation which was a classical example of acute malignant endocarditis. Was this vegetation singled out by some secondary or superimposed infection, and all the other lesions left to the rheumatic infection ?

A severe aortic and mitral lesion developing in the same attack of rheumatism always suggests to me a warning of malignancy, and my experience supports my fear, for I have collected a number of these cases which died eventually of this form of endocarditis.[10] Within three months in 1932 I had four such cases in young people with this double lesion dating from childhood. One of them, a young ward sister, developed multiple painful arthritis with effusion reacting to salicylates. It was an illness precisely resembling acute rheumatism, but her temperature remained raised, and looking at her finger-tips I saw the fatal Osler's spots.

In some respects the most remarkable case of organic heart disease in childhood met with in my experience was the following. The mother of an infant had an attack of acute rheumatism during late pregnancy from which she recovered, but her child was born deeply cyanosed, and died on the third day. The post-mortem examination showed an extreme endocarditis of the mitral valve with fungating vegetations which contained numerous streptococci. There was no evidence of a suppurative lesion at the umbilicus or elsewhere,

and even if this had been found the fact that the child was born deeply cyanosed and had no congenital malformation of the heart would have been difficult to interpret in any way other than as the result of an ante-natal infection.

These, then, are some of the reasons upon which I base my view that there is a rheumatic malignant endocarditis. The explanation is not a complete one: it cannot be with the present uncertainty of medical thought, but I seem to see some further light dawning in favour of the view. For many years I have believed that the rheumatic poisons were so subtle that they built themselves into the living cells of the body. This is, I admit, a primitive thought, but somewhat supported by the recent studies of allergy. This term I use with the utmost caution, and say at once that when a child develops a first attack of acute rheumatism with general carditis that is not in my opinion an allergic condition. Acute rheumatism is, I hold, not allergic, though I believe it to have in its life history an element of allergy, and that the body when once attacked by acute rheumatism may become sensitized to that streptococcal infection. Then may result a failure of the tissue cells and leucocytes in a damaged cardiac valve to destroy lurking streptococci, and a failure of the entire system to react against the fatal focus lying directly in the track of the blood-stream. There is no other rheumatic lesion that lies in such a relation to the circulation, but I believe a pericarditis may also be malignant in type, though by its anatomical arrangement it cannot produce a condition comparable to that of a malignant endocarditis. The problem of malignant endocarditis in the rheumatic is, I hold, not to be solved by an easy assumption of a super-imposed infection, but requires a more prolonged study of rheumatic

endocarditis, of the disease "rheumatism," and also of the streptococcus.

In touching upon allergy I do no more than show that I am alive to that aspect, and I should also wish you to know that I have also in mind the recent work of Professor Hadfield and his colleagues[13] upon the lysis of fibrin by hæmolytic streptococci, which is of deep interest.

A Transitional Type of Rheumatic Endocarditis.

In January, 1934, a boy of six years illustrated another feature in this problem. This boy was admitted with pericarditis, mediastinitis, endocarditis of the mitral and aortic valves, and numerous subcutaneous nodules. It was not his first attack of rheumatism, and the physical signs pointed to an adherent pericardium. The acute attack died down and he became a very cheery boy, yet in spite of this began to lose ground slowly, and though for weeks before his death there was no fever, he wasted, his pulse remained persistently rapid, and his face became pinched and livid. In this toxic condition he died. From previous experience I demonstrated upon him as a malignant rheumatic endocarditis of the transitional type.

The autopsy showed the adherent pericardium and mediastinitis. The aortic and mitral valves showed vegetations, those on the aortic of the usual rheumatic type, those on the mitral larger, and resembling those small vegetations you see in some areas of a frank malignant endocarditis or on the mural aspect of the endocardium.

Such cases Dr. Paine and I described in 1912,[11] and republished in "Researches on Rheumatism." I think it is important that the diagnosis was made in this case during life and confirmed after death, and such a case provides, in my opinion, a missing link in our problem which may be easily overlooked. There is the clinical "simple" rheumatic endocarditis, there is the malignant type occurring in a rheumatic subject, and joining these a rheumatic type malignant in its

toxicity and persistence, but with vegetations which if looked upon as a case of acute rheumatism would be called large, or if looked upon as a case of malignant endocarditis would be called small. I have dwelt so long upon this subject because I feel there is some hope that, if my view is correct, we may eventually prevent the rheumatic form of malignant endocarditis, and at least this is a line of approach less hopeless than the present one of negativism. It has received of late increasing support upon the Continent, and I need only mention in proof of this the names of Professors Talaliev, Schotmüller, Herschfield, Lang, Orth and Hasincamf.

TUBERCULOUS HEART DISEASE.

Tuberculous heart disease has been in my experience uncommon among children in London. I put aside general miliary tuberculosis, for the cardiac lesions are so minute as to be of no practical importance as compared with the pulmonary and meningeal lesions. I have, however, seen sufficient of this heart disease to get a clear idea of the general clinical features in the child. Tuberculous pericarditis is the lesion most commonly met with, but valvular heart disease may occur with it, or independently of it, though I believe myself correct in stating valvular lesions are rare, and these when chronic may show much calcification, as I have seen. It is very probable that we shall mistake the early signs of tuberculous pericarditis for rheumatic pericarditis, because the rheumatic form is so much the more frequent. Nevertheless, I think we may be suspicious when there are no other signs of rheumatism, although we must admit that a primary cardiac rheumatism—

that is, a rheumatism of the heart with no arthritic pains, no joint swellings and no chorea—may certainly occur and be severe enough even to prove fatal. Again, I think we may be also suspicious if there is pericarditis without detectable valvular disease, for this in rheumatism is unusual, but though it is unusual this again certainly occurs clinically, and on the other hand, as already mentioned, tuberculous endocarditis is sometimes present. Again, the Mantoux test may help us in a suspicious case.

The tuberculous form of pericarditis takes two different clinical courses, with the usual reservation of the clinician that there may be transitional cases. In the first place it may cause a large serous or blood-stained effusion for which paracentesis of the pericardium may have to be undertaken not once only but sometimes twice or thrice. In this effusion the tubercle bacilli may be found in some cases in great numbers, but in others they cannot be found, and the diagnosis is confirmed perhaps by the infection of a guinea-pig, although we may be morally certain from the character of the fluid and the excess of lymphocytes as to its nature.

Paracentesis of the pericardium for a child under twelve years of age on account of rheumatic pericarditis is so rarely indicated in London that I have not had a case since the War. In spite, then, of difficulties we generally arrive at the correct diagnosis of the nature of tuberculous pericarditis when we are driven to paracentesis. Such cases may die from the acute condition or recover, possibly to die later from a general tuberculosis.

Very different and very remarkable is the plastic type of tuberculous pericarditis. I have had

opportunities of following such cases from the commencement to the end.

The pericarditis is a part of a general subacute tuberculosis of the serous membranes, and may be the first membrane attacked or appear later when a tuberculous pleurisy has been detected and subsided under treatment.

When it is the first event there is the usual difficulty of excluding rheumatic pericarditis upon which I have already dwelt. The pericardial friction is indistinguishable, but there is not the cardiac dilatation met with in rheumatism, and as there is probably no valvular disease you feel at the back of your mind that the case is an unusual one, but may get no farther than that. Slowly the pericarditis subsides after some weeks of irregular fever, and the child may leave the hospital apparently cured, or recovery may be interrupted by a pleurisy on the left side, or either pleura may be singly affected. There is usually no effusion with this pleurisy, but the subsidence is slow and the signs of a thickened pleura follow. Eventually the child partially recovers, and a year or two later, during which time the health has been indifferent, is brought to hospital for ascites. It may happen that the early stage I have described may not have been under observation, and then we are confronted by a child with a mysterious ascites. The immediate diagnosis is generally tuberculous peritonitis, but this is unsettled by the much enlarged liver and spleen and the insistence of signs of cardiac weakness.

Examination of the heart requires minute care, for the solution of the problem lies there. The cardiac dullness may be but little increased, but

in a classical case it seems to be unusually massive and wooden, and on listening to the heart the sounds are muffled and feeble, particularly the first sound. Again, there will probably be no valvular murmur. Examination of the chest may discover a diminution of the air entry in one or both axillæ and a loss of resonance on percussion, and a sense of resistance which suggests a thickened pleura.

The pathology of the condition explains the cardiac signs. The pericardium is greatly thickened with possibly calcareous plaques embedded in it, and the heart is of small size and strangled by the thick pericardium. The ascites gains ground, and many repeated paracenteses of the abdomen are needed. In this pitiable state the child may survive for a year or more, and then die from exhaustion, or there may be the involvement of another serous membrane which may give rise to symptoms that are liable to be most puzzling if the case is not understood. Ptosis may develop with headache, retraction of the neck, vomiting and convulsions. The serous membrane of the brain has at last been attacked, and death speedily follows. At the post-mortem examination the peritoneum, though possibly thickened from prolonged ascites, may not show any tubercle. Not all cases die from meningitis, but the above is the classical course which I have followed with care through every phase in three cases. Only once have I seen a multiple serositis with repeated ascites in the acute rheumatism of childhood. In this case there was an adherent pericardium and chronic rheumatic peritonitis, in the early phase of which I detected peritoneal friction over the liver and

spleen. This child I followed for more than two years, and an attempt to deal with the ascites surgically proved fatal.

A case of tuberculous pericarditis of this type[12] was the occasion for the first example of Brauer's operation for cardiolysis published in this country, though Allingham had already undertaken this procedure. In my case the patient was a boy of 13, who even when he walked about the hospital ward developed œdema of the legs and breathlessness. There was no valvular disease, but massive precordial dullness and remarkable diastolic recession of the intercostal spaces on the left side over a wide area. I had thought the case was rheumatic. Mr. Trotter, on opening the chest, found a large tough adhesion shackling to the parietes the area around the heart's impulse. It was a very remarkable sight to see the heart spring back when this was severed.

Recovery was remarkable, and for two summers the boy was well enough to act as a scorer for his school cricket club. Then he developed a left pleurisy with effusion and this was tuberculous. Later he died of pulmonary tuberculosis, but no autopsy was allowed.

I have never advised this operation for a rheumatic child, either because I was uncertain of my diagnosis or because there was in my opinion too extensive valvular disease, or perhaps because I was not sufficiently courageous. Others, however, have reported successes.

PNEUMOCOCCAL HEART DISEASE.

In London pneumococcal heart disease in childhood is in my opinion infrequent, though it cannot be called rare, and this infection usually attacks the

pericardium. I can only recall one case of acute pneumococcal endocarditis in an infant. This child of eighteen months had an acute apical pneumonia with high fever, and developed a loud aortic systolic murmur. After death there was found a large fungating vegetation on one of the aortic cusps which gave a pure culture of the pneumococcus lanceolatus.

Pneumococcal pericarditis, which we know is more frequent in children of under four years than at any other age in childhood, may occur at all ages.

Undoubtedly this pericarditis may spread direct from a pneumococcal pleurisy, and in former days, when empyemata were not dealt with surgically, the end was not uncommonly suppurative pericarditis. It is, however, generally an independent infection of the pericardium, and this was strikingly illustrated in one of my cases in which the left upper lobe was solid with pneumonia, but neither its pleura abutting on the heart nor the corresponding external surface of the pericardium was implicated, although there was extensive internal pneumococcal pericarditis. This form may, we know, be accompanied by a copious purulent effusion, and from time to time we see and read of cases which have been successfully operated upon. In some of the cases with great effusion I have known the heart sounds completely disappear. The prognosis is always serious.

I would again venture to dwell upon this point, the rarity of malignant endocarditis in the pneumococcal infections of childhood.

Very different are the more frequent cases in early childhood, and I think the diagnosis is one of the most difficult in heart disease. Often

enough, it is true, the diagnosis is of no practical importance, for opening the pericardium could be of no service when, after death, you find only a thin plaster of pus, and the child has evidently died from a severe pneumonia with possibly empyema, peritonitis and meningitis. Every clinician is, however, disturbed when he finds a lesion at autopsy which he has not even suspected during life, and asks himself the reasons for the failure. One reason, and a formidable one, is the usual absence of pericardial friction. It is very interesting to realize the difficulty there is in the diagnosis of pericarditis when there is no pericardial friction, particularly when there are also no endocarditis, and no great dilatation of the heart, as in rheumatism, to attract your attention. I have some interesting figures upon these points.

From a study of 100 fatal examples in childhood I found that about 80 per cent. of cases of pyo-pericardium occur under four years, and about 60 per cent. at three years and under. There was no practical difference in the number of cases that were associated with an empyema on the left side as compared with the right or with empyemata on both sides. A large purulent effusion was exceptional, and in only about 25 per cent. did the amount of pus found in the pericardium exceed three ounces.

At one time I had in my mind that pneumococcal pericarditis was rapidly fatal, but from my study of these cases I was disabused of this view, for in seventeen there was evidence of a much thickened pericardium and of much attempt at organization of the exudation, changes which must have taken months to have developed. I only found a record of pericardial friction in four cases, but this

cannot be accepted as more than a proof of its infrequence.

Then, again, another difficulty in the diagnosis of this form of pericarditis is that the child is generally very ill with respiratory lesions, and when the temperature remains high or irregular we naturally search for an empyema, and sometimes when completely puzzled fall into the trap of changing the diagnosis to one of acute tuberculosis.

Fainting attacks, rapid and changing rates of pulse and irregular spikes of fever have been observed in these cases, but I have not found them at all constant, and certainly fainting attacks may not occur. Another difficulty is one which is irritating. You see a case, and vow that you will not miss the next one; but the interval of time before you see the next case is just so long that you forget your vow and fail again. Fortunately, as I have said, failure may be of little practical importance, and those cases which are urgent, those with a considerable effusion, give those clinical signs that are recorded in every text-book and radiography also comes to our assistance.

I end this lecture in the spirit of Research—a research to link the emotional with the physical heart. Physicians of experience have frequently noticed that when a case of organic heart disease—a case, for example, of a hard-working artisan—is nearing the end, and I have noticed this particularly with cases of aortic regurgitation, there comes upon him a curious state in which, however ill, he insists upon going home, and his faithful wife, in spite of anxiety and danger, welcomes him back.

Some here will know what it means to work year in year out at a subject until Time touches him on the shoulder. Those will then understand what

pleasure it gave me when I received from this University—which casts its influence over a part of the country my family have been connected with for over 150 years—the invitation to give this lecture, a welcome to the lad of seventeen to come back and tell them some of his work and experiences. This invitation has greatly pleased me, for it is the first greeting I have received from our own universities, though not the first from our Dominions and America. I have, then, asked your Faculty of Medicine to accept as a thank-offering the original manuscripts of the two papers which I have thought to be the most important that I wrote with my late colleague Dr. A. Paine on the subject of acute rheumatism, and with them our collected papers as published in "Researches on Rheumatism."

These are now naturally documents of the past, but it is possible that in future days these manuscripts may be of interest to the medical historian and to this University as the work of two Somersetshire men.

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ABSCESS OF THE BRAIN,

WITH REPORTS OF TWO CASES.

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ABSCESS of the brain is a condition fortunately not very common, but none the less one very important to recognize. At the present time the mortality is high, and at least in part this is due to the fact that a large proportion of the cases are not recognized or are recognized only in the late stages, though even of those cases diagnosed and treated early a larger proportion die than should be the case.

ÆTIOLOGY.

The initial infection may reach the brain in several ways.

Trauma.—As a result of trauma. (1) Direct, as in penetrating wounds; (2) indirect, as with fracture of the base of the skull. We need not further consider these cases except to remark that (*a*) an abscess may arise months or years after the original injury, and (*b*) in some cases of abscess arising apparently by direct extension there is a history of trauma, such as a blow on the head.

Hæmatogenous.—By the blood-stream. (1) Arterial. (*a*) In acute pyæmia multiple abscesses in the brain

may occur. (*b*) Arterial embolism may lead to *metastatic abscess*, usually single, where any chronic infective process is present, for example osteomyelitis, malignant endocarditis, even dental or tonsillar infection: the only important group of cases are those following lung suppuration, particularly bronchiectasis. (2) Venous septic thrombosis may lead to the extension into the brain of infective processes in the neighbourhood, and probably accounts for some of the abscesses that appear to have resulted from direct extension: the only other important group of this class is that in which the veins of the under surface of the frontal lobe carry infection via the cribriform plate and the ethmoidal vein from the angular vein, as may occur following a boil on the face or eyelid.

Direct Extension. — By direct extension from suppuration in the immediate vicinity, that is, usually, in the sinuses of the nose and particularly in the mastoid process. This last class is numerically by far the most important. The actual pathology appears to be: inflammation of the dura, inflammation of underlying arachnoid, matting together of dura, arachnoid and adjacent brain, extension of infection into the brain along the perivascular spaces. As has been said, in some of these cases the venous path is responsible; and occasionally a metastatic abscess will arise from ear disease by the arterial route, in a part of the brain remote from the original focus.

The very great majority of abscesses secondary to sinus or aural disease arise in the course of chronic suppuration (as opposed to acute), and where the ear is concerned nearly always there is cholesteatoma formation. This may in part explain why brain abscess is uncommon in children, but not entirely why

males are rather more often affected than females. The infecting organism is nearly always a streptococcus or a pneumococcus, in about equal ratio, but other organisms sometimes occur, and mixed infections are not uncommon.

CLINICAL AND PATHOLOGICAL TYPES.

Once infection has occurred the course of the disease is governed by the relative virulence of the infection and the effectiveness of the response. Although no hard and fast line can be drawn, it is possible both clinically and pathologically to recognize three types.

Suppurative Encephalitis.—When the infection is of high virulence it produces a condition comparable rather to cellulitis than to abscess. In the centre of the affected area is a core of dead brain tissue; round this a zone of engorged brain in which organisms and pus cells can be found, but with relatively little tissue reaction; and outside this again a zone of œdema. Such a condition is sometimes described as "suppurative encephalitis" to emphasize the distinction from true abscess. Clinically these cases tend to have an abrupt onset, with headache, high fever, perhaps a rigor or convulsion, and to suggest the occurrence of meningitis. Localizing signs may be seen early, but are soon merged in the profound stupor from involvement of a large part of the brain in the œdema: although the results of operation are poor, a few recoveries are obtained.

Sub-Acute Abscess.—With an infection of lower virulence an actual abscess is formed, that is, a space filled with pus and deliquescent brain substance, the walls formed of necrotic brain tissue; round this is a zone of active inflammation, which gradually forms

a belt of granulation tissue; and outside this zones of engorged brain and œdema. In the course of time the granulation tissue organizes, and the abscess cavity, the size of which may increase very slowly or not at all, comes to be surrounded by a fibrous covering which is intimately joined to the surrounding brain, lined with a pyogenic membrane. This is the commonest variety of abscess, the clinical features of which are considered later.

"*Tumour.*"—At the extreme other end of the scale we find cases of very gradual evolution, where the clinical features are those of tumour formation rather than of abscess. Such a diagnosis having been made, the "tumour" should be removed complete in its capsule: when this is done, and the "tumour" cut across, it is found to consist of a thick mass of fibrous tissue, in the centre of which may be quite a small cavity containing pus, perhaps sterile.

CLINICAL COURSE.

The clinical course of a "typical" brain abscess can be divided into three stages, although the division must be regarded as somewhat elastic, signs belonging to one sometimes appearing in another. The onset is heralded as a rule by signs of renewed activity in the adjacent suppurative focus; for example, an ear that has for years been painlessly discharging may become painful, while the discharge ceases.

Invasion.—The stage of invasion. In the majority of cases the onset is insidious. The patient complains of severe headache and of feeling very ill, but there may be nothing more to attract attention. The headache is usually "all over the head," but may sometimes show the special features described below. In children there may be no complaint of headache.

The temperature is somewhat raised, 100–101° F being common, and the pulse-rate is correspondingly increased. Occasionally there may be nausea, vague giddiness, or a feeling of "chill." In some cases, however, the onset is more dramatic, with a rigor and vomiting, or even convulsions—particularly with metastatic abscess. Or signs such as are described under the third stage (of compression) may be observed. Finally, a patient who has not previously complained may suddenly become comatose without warning.

Latent Stage.—The latent stage. Following the invasion, the patient may recover to a considerable extent, but not completely. The headache recurs, especially on lying down, or on any sudden change of position. The complexion takes on a dull, muddy tint. Appetite is poor or capricious, all food may taste unpleasant, and there may be considerable wasting—this is occasionally extreme. The tongue is furred and constipation may occur: nausea or even vomiting sometimes appear at this stage. The temperature is normal, as a rule, but shows some irregularity. If it is taken every half-hour, an occasional spike of high temperature, quite transient, may be observed. The pulse may show also a transient phase of slowing, 46 or 50 being counted for an hour or so only. The greatest changes, however, are mental, and often the relatives are the first to point out that "he isn't a bit like himself." The patient is irritable, cannot settle to anything like reading or writing and cannot concentrate; he may have periods when he is dull and lethargic; children are fretful, and will not play with their toys; and periods may occur when the changes of the next stage are seen. As a rule no useful information can be obtained by examining the eyes at this stage, but leucocytosis

occurs and a lumbar puncture will show characteristic changes (p. 237). This stage may last four or five days or even several weeks, and merges gradually into the next.

Compression.—The stage of compression. This is the stage in which are seen the "classical" signs of brain abscess; but we should certainly not wait for it to develop if there is any possibility of making the diagnosis earlier. Although occasionally a brain abscess may reach a very large size, as a rule it is to the surrounding œdema that increase of intra-cranial pressure must be ascribed. The symptoms and signs are (*a*) those of cerebral compression and (*b*) localizing signs. *All* the signs and symptoms of this stage tend to vary in intensity from time to time, sometimes rather rapidly.

The mental state becomes still more obviously abnormal: the patient is drowsy, and may go off suddenly to sleep. He cerebrates slowly, and may even appear deaf (be sure that he is *not* deaf); on being asked a question several seconds or even minutes may elapse before he produces the answer, though the answer when it does come is correct. (In the mental changes seen in the worst cases of toxæmia from nasal sinusitis exactly the same symptom may appear.) Irritability, wasting, and the muddy complexion are still more marked. The temperature may be raised, but as a rule it tends to be subnormal, although irregular with an occasional rise, and the pulse to be slow—or at least slow in relation to the temperature. As pressure increases the pulse may show a somewhat rare, special character; that is, with inspiration it is rather slow, but with expiration it becomes very much slower for a few beats—for example, during inspiration 60, during expiration 44. Vomiting occurs,

which is not specially related to food, and which sometimes shows a sudden "projectile" character. Headache is more severe and constant, though showing variations in intensity; it is often worse on the side of the lesion. After movement the patient may complain that "my head feels as if it is bursting," and this feeling may herald an attack of vomiting. Some degree of rigidity of the neck may be found, even when the abscess is supra-tentorial.

Eye Signs. — Photophobia may be noticed, or slight, fine nystagmus of labyrinthine type, if the eyes are turned well out to one side or the other—even though neither labyrinth nor cerebellum is affected: nystagmus due to one of these causes is discussed later (p. 242). Diplopia may occur, usually from paralysis of the external rectus; unfortunately this occurs in so many conditions that it is of little help in diagnosis, and it may not even indicate the side of the brain affected. Rarely diplopia is due to third-nerve paralysis, most often with cerebellar abscess, sometimes with a frontal lobe lesion, least frequently with a temporo-sphenoidal abscess. The pupil on the side of the lesion may be dilated, or may react more sluggishly than the other. In a proportion of cases papillœdema will be observed, particularly where the posterior fossa is involved: it tends to be more pronounced on the side of the lesion, but it is by no means constantly seen, even with an abscess of considerable size and duration. With severe papillœdema vision may be impaired, sometimes permanently.

Lumbar Puncture.—It cannot be too often emphasized that there is a definite risk in performing lumbar puncture when a brain (especially a cerebellar) abscess is present. In any case not more than 2 c.c.

should be withdrawn. The tests that give information of value are the cell-count, the differential cell-count, estimation of chlorides, and culture of the fluid; the estimation of glucose is of secondary importance in these cases, since the value of the examination is largely to distinguish early cases of abscess from meningitis, septic, meningococcal or tuberculous.

One may summarize the findings thus:—[1]

Pressure.—Normal 100—150 mm.=not more than two drops per second from the needle. Slightly increased in straining, stertor, meningismus, encephalitis, early septic meningitis or abscess; greatly increased in all other meningitis, tumour, late brain abscess.

Appearance.—Normal = water-clear. Clear or faint opalescence with brain (or extra-dural) abscess, early meningitis (any variety); turbid with established septic or meningococcal meningitis, or "leaking" brain abscess. 500 lymphocytes per c.mm. may produce only faintest opalescence, 200 polymorphs usually give definite turbidity.

Cells.—Normal 0—4 per c.mm. all mononuclears. With brain abscess *any* variation from this may be seen, the most usual being 10—30 cells per c.mm., of which 10—30 per cent. are polymorphs. If all mononuclears, probably tubercle, syphilis or tumour, encephalitis, poliomyelitis, etc. If polymorphs are 10—70 per cent. (10—500 per c.mm.) may be tubercle, syphilis, early septic meningitis or influenza. If polymorphs 70—90 per cent. (100—5,000 per c.mm.) leaking brain abscess, meningitis.

Bacteria.—Few in tubercle, meningococcal meningitis (at first). Streptococci, pneumococci mainly extra-cellular. If they grow on culture prognosis bad; turbid fluid with numerous dead bacteria and normal chlorides suggests leaking abscess.

Wassermann.—Positive reaction may occur with tumour or abscess apart from syphilis.

Glucose.—Normal 0·045—0·08 per cent.; slight *increase* with encephalitis, abscess; diminished with tuberculous or early septic meningitis; absent in septic or meningococcal meningitis or leaking abscess.

Chloride.—Normal 0·72—0·75 per cent., and with abscess; diminished in meningitis, typhoid, pneumonia; below 0·65 per cent.=tuberculous meningitis.

Other methods of investigation are skiagraphy, by which occasionally an abscess may be seen, ventricle puncture, or ventriculography (skiagraphy after injecting air into ventricles).

Termination.—If the condition persists unrelieved death will occur, either with deepening coma or respiratory paralysis from pressure, or from meningitis, or from rupture of the abscess into a ventricle (very high temperature, unconsciousness, stertor).

LOCALIZING SIGNS.

The localizing signs of brain abscess are often few and transient. The abscess itself very rarely involves a part of the brain where lesions have characteristic signs; it is from involvement of surrounding parts in œdema (the extent of which may change rapidly) that signs occur. And therefore it is of the greatest importance to observe the patient closely and often in order not to miss these. The surest guide as a rule is the discovery of the original focus of infection; the abscess is seldom more than half an inch deep in the brain from this,* unless a metastatic abscess is present. Tenderness on percussion over the abscess is a valuable sign when observed: it is infrequent, and difficult to distinguish from tenderness due to the original focus.

Frontal Lobe.—Where the nasal sinuses are the starting-point the abscess is to be sought in the lower part of the frontal lobe: only when both sides show sinusitis will there be difficulty in deciding where to operate, and here there may be some help from the position of the headache. But frontal lobe abscesses are notoriously "silent," and may exist for long

* It may, of course, lie as deep as two inches from the external surface.

without even being suspected. One feature may give rise to suspicion—the patient is unduly optimistic, and although visibly getting worse is convinced that he is improving. Ocular palsies may sometimes help in the diagnosis.

Temporo-sphenoidal Lobe. — When the ear is to blame, even when we have decided which ear, we are no nearer knowing whether the abscess is in the temporo-sphenoidal lobe or in the cerebellum. The former is at least twice as common as the latter, but much more likely to be without localizing signs. These when they occur are most often due to œdema affecting the motor cortex above, or the fibres passing down from this through the internal capsule, with consequent *contra-lateral* weakness. Where the motor cortex is affected the lower part of the face is most likely to be affected, the corner of the mouth being weak (especially when at rest or on involuntary movement); the arm also may be affected, but the leg usually escapes. The reverse occurs if the motor tracts are involved at the internal capsule, the leg being most likely to suffer, the arm less so, while the face escapes. In the latter case the contra-lateral knee-jerk will be absent in the early stages; later it is brisker than normal, and the plantar reflex may be extensor, but the leg is "weak." Weakness may, of course, go on to paralysis.

When the left side is involved (extremely rarely the right) the "naming test" may be positive: the patient is shown a number of common objects and asked to name them one after another. If the test is positive he may name eight or ten correctly, and then find himself unable to name the next, although he knows quite well what the object is, and can pick out the right name from a list. Thus he may say:

"That's what you cut up your meat with; it's made of iron," etc., but he cannot remember that it is "a knife."

A sign rarely seen, but valuable when present, is a defect in the upper quadrant of the visual fields of both eyes on the side opposite the lesion, from involvement of the optic radiation as it sweeps back towards the calcarine fissure. Another rare sign is complaint of persistent bad smell (or taste), unrelated to food: this may indicate a lesion far forward in the temporo-sphenoidal lobe (or it may give the clue to unsuspected sinusitis).

Cerebellum.—When the cerebellum is involved the chances of complete absence of localizing signs are less. In the first place, nearly all cerebellar abscesses are secondary to lateral sinus thrombosis or to labyrinthitis—in about equal numbers—and lie in the region of the cerebellum nearest the structure concerned. Again, increase of intra-cranial pressure is earlier than with supra-tentorial abscesses, and so tend to be vomiting, bradycardia, neck rigidity and papillœdema. But the cerebellar cortex is "silent," and localizing signs depend on the deeper nuclei being affected. Signs are most likely to appear in the arm of the *same side*, and to a less extent in the leg; the face escapes. The limb in flaccid and weak and the tendon reflexes diminished; inco-ordination is present, and movements in which several joints are affected may be performed in stages, one joint being moved at a time; dysdiadochokinesis may be discovered. If the patient pulls against resistance, and the resistance is suddenly withdrawn, his hand flies away up, instead of being arrested in a short distance as normally. Nystagmus is present, and Rombergism, the patient tending to fall towards the side of the disease. Since

several signs closely resemble those of labyrinthitis, it will be convenient to contrast the two conditions, remembering always that both may be present at once.

Cerebellar Abscess.	*Labyrinthitis.*
Onset. — Gradual, symptoms increasing in intensity during several days. Vertigo may be mild. Vomiting often late, sudden, related to headache rather than food, perhaps without nausea.	Abrupt, with intense vertigo, vomiting, nystagmus. Symptoms tend to diminish in a day or two, but recur on movement. Vomiting related to movement, always with nausea.
Nystagmus.—Of cerebellar type, irregular, coarse, horizontal, quick component directed to side of disease, looking toward which makes nystagmus worse : on looking to healthy side slight fine nystagmus of " labyrinth " type may be seen.	Of labyrinthine type, fine, regular, mixed rotatory and horizontal, quick component directed to healthy side, looking toward which makes it worse ; lessened or abolished on looking to diseased side.
Falling.—Tends to fall toward the diseased SIDE, no matter what the position of the head.	Tends to fall in direction of diseased EAR. Thus, right ear diseased, head turned to right, tends to fall backward, head turned to left, falls forward.
Past-pointing.* — The arm of the *diseased side only* deviates outward.	*Both* arms tend to deviate toward side of diseased ear.
Ear. — If deaf, deafness of " EAR " type. Tuning-fork on mastoid heard better than on normal mastoid, on vertex heard better in diseased ear (if other ear healthy). Syringing ear with hot water (110°) may bring on labyrinthine nystagmus to same side ; with cold water, to opposite side.	(Probably) complete (nerve) deafness to all sounds ; tuning-fork on vertex or mastoid heard only in opposite ear. Syringing ear with either hot or cold water produces no effect at all.

* The patient sits with eyes shut and arm extended forward, index finger touching fixed object ; raises hand above head, then brings it down in attempt to touch object again.

DIAGNOSIS.

When all has been said, it must be admitted that the diagnosis of brain abscess in an early stage is fraught with difficulty, mainly because the symptoms are so little characteristic: "influenza" is the catch. Possibly the condition may be remembered if a patient without very definite signs complains of chilliness, nausea, or of feeling much more ill than one would expect, especially if he has chronic otitis media or bronchiectasis. When indefinite cerebral symptoms are present tuberculous meningitis or encephalitis may be suggested. To misinterpret cerebral signs in malignant endocarditis is perhaps of little practical significance. If the patient is comatose when first seen one has to consider uræmia, diabetes, alcohol and drugs: cerebral hæmorrhage in the posterior fossa may produce slight fever, pain and tenderness, rigidity of neck, nystagmus, (nerve) deafness and (permanent) changes in the labyrinthine reflexes. When there have been symptoms pointing to activity in old ear disease any intracranial complication is likely to be lateral sinus thrombosis, labyrinthitis, septic meningitis or brain abscess. A rigor in a subject of otitis media is nearly always evidence of thrombosis, a second may be considered conclusive (exclude malaria). Meningitis is of sudden onset, with very severe headache, and might lead to a mistake in diagnosis: it tends to get rapidly worse, and should be excluded at once, since it is one of the few surgical conditions where every hour makes a difference to the prognosis. Brain abscess is seldom so urgent as this, unless advice is sought only in the later stages, and then one may hope to observe at least some of the signs and symptoms described above; but a clear and complete picture is rare.

TREATMENT.

There has been some discussion whether it is better to operate as soon as a diagnosis can be made with reasonable certainty, or to wait "until the abscess is walled off." My own views are definitely in favour of early operation: the risks the patient runs while waiting for a favourable issue are so much greater than those of exploration of the brain in proper hands. The details of operative technique we need not discuss.[2] The principles are: (1) large skin and muscle flap, wide resection of bone; (2) small opening in the dura, and careful and systematic exploration of the brain with a blunt trocar; (3) the abscess being found, fix a small drainage tube without removing the cannula; (4) apply a large dressing, and if the general condition is satisfactory do not touch the brain for at least a week—no probing, no irrigations, no "breaking down loculi." Recoveries have been recorded even after rupture of an abscess into a ventricle or cessation of respiration from medullary compression.

Case 1.* Abscess of left temporosphenoidal lobe secondary to otitis media; operation; recovery.

William B., aged 60. 24th June, 1934, complained of pain in left ear for fourteen days. The ear has run ever since he was a small boy, ceased six months back, discharged again five days ago, but without the relief of pain that he expected. Headache severe "all over." Definite rigidity of neck, moving head brings on pain in vertex, drowsy all the time. Pain and tenderness behind left ear, no redness or œdema. I saw him first on 25th, when rigidity of neck was barely noticeable. He was restless, throwing himself about and groaning with pain in left half of head; constantly nauseated, not vomiting—but he had vomited five times earlier in the day, and also on 23rd; no special character in vomiting. Temp. 96°; resp. 22. The pulse showed great variation in rate; with inspiration about 80, with expiration 48 or 50, but only for four to six beats. Average rate had been at 8.30 a.m. 84, at 9.30 a.m. 60, at 1 p.m.

88, at 2 p.m. as just described. He was rather confused, but understood simple questions, named objects correctly. Past-pointing could not be tested, vestibule not tested, no nystagmus, bone conduction increased. Knee-jerks absent, plantars flexor. Diagnosis: temporosphenoidal abscess.

Immediate operation: left radical mastoid operation. Bone cortex dense, acellular type, antrum full of mass of cholesteatoma which had "dissected away" all cancellous bone; no trace of ossicles found. Skull opened immediately above attic and antrum with nibbling forceps, brain explored through small dural incision with P. Watson-Williams's sphenoidal sinus cannula.[2] The first exploration reached an abscess 1 cm. above floor of middle fossa and 2 cm. from external surface of brain; about 15 c.c. of thick pus came out through cannula and afterwards through tube. When the dura was first opened the brain bulged tensely out, but pulsated well. A narrow drainage tube previously threaded over the cannula was slipped down to the tip without removing the cannula from the abscess, and secured by suture to the dura before the cannula was withdrawn: the pus grew streptococci on culture. The brain was dressed with strips of gauze soaked in glycerine, and the anterior part of the wound closed.

Next day (26th) very much better, takes fluids well, no headache, no vomit. 27th, pulse 88, temp. just above normal in evening, takes soft solids, quiet, intelligent (at least, his "normal"). 30th, whole ward stinks; outer dressing of sphagnum moss fibre, inner dressing not changed. 5th July, general condition very good; brain tube changed for first time (11th day); not irrigated. 3rd August, plastic closure of wound. Convalescence uneventful. 3rd September, he finds some difficulty in remembering *names* of things: left tinnitus, and some vertigo if he turns quickly. 18th October, very satisfactory; still occasional vertigo.

Case 2.* Abscess of right temporosphenoidal lobe secondary to otitis media; operation; recovery.

William G., aged 43. Came 5th July, 1934, complaining of severe frontal headache for one week, right aural discharge six days (he said not previously ever !), drowsiness and vomiting noted during a few hours. Temp. 100°, pulse 72, resp. 30. Seen 11 p.m. Pale, drowsy but quite sensible, prefers to lie

* These cases shown at the meeting of the Bristol Medico-Chirurgical Society, Dec. 12th, 1934.

in bed on left side, skin hot and dry, pulse full and regular, tongue furred, dry. Neck rigid but not true "retraction"; groans with pain in the forehead, especially on any movement of head. Names objects correctly. Right ear: free purulent discharge, central perforation, no pain, tenderness or swelling; bone conduction increased, Weber to right, no nystagmus or vertigo. Vomited several times earlier in the day apart from food, complete anorexia. Slight weakness of left corner of mouth. Knee-jerks, right weak, left normal; plantars, flexor, right weak (? possibly =left reflexes+). Kernig's sign absent. No inco-ordination or ataxia (could not test walking). Lumbar puncture, pressure much increased, fluid cloudy; numerous polymorph. pus cells, no organisms seen (culture sterile), glucose absent. Diagnosis: cerebral abscess; 3 c.c. concentrated antistreptococcal serum intrathecally, 3 c.c. intramuscularly.

Immediate operation. Right radical mastoid. Cortex thick and dense, antrum full of cholesteatoma, pus offensive.

Dura of middle fossa had been exposed by erosion of bone above posterior part of antrum, and reddened there. Free exposure of dura by subtemporal route (as in Case 1);[2] dura pulsated well. Exploration of brain, abscess entered at first venture 2·5 c.m. from surface; very offensive "watery-curdy" pus escaped cannula (culture later reported "streptococcocci and leptothrix"). Drainage tube passed and secured, cannula removed, glycerine dressings.

6th July. General condition good, pulse 68. No headache, taking fluids well, no vomit. Optic discs normal. Later: pulse still slow (60—70), smell very offensive, sphagnum moss dressings.

13th July. Brain dressed and drainage tube changed for first time (8th day), no irrigation. Still gets headache on movement.

27th July. Up in chair; no headache.

14th August. To convalescent home; pulse now normal.

25th October, 1934. Very satisfactory recovery. No symptoms; back at work.

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CEREBRAL DERMOID.

BY

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CEREBRAL dermoids are of sufficient rarity to call for record in each instance.

The classical work on the subject is that of Bostroem,[1] published in 1897. More recently the subject of epidermoids and cholesteatomata has been dealt with by Percival Bailey and Horrax in America,[2] and by Critchley and Ferguson[3] in this country, but the rarer dermoids have received comparatively little attention. Brock and Klenke,[4] however, published in 1931 an account of a case of cerebral dermoid, and included in their paper a full and clear summary of all the recorded cases up to that date with a critical survey of their pathology.

The rarity of these growths is shown by the fact that until 1934 only 39 cases had been reported, and we have only been able to find details of three or four described since that time. In Cushing's series of 1,936 brain tumours only three were

dermoids, and in the New York Neurological Institute a series of 450 verified tumours included but two dermoids.

Epidermoids are two or three times as common as dermoids, and according to Bostroem are embryonically older, arising in the first two or three weeks of fœtal life, whereas dermoids do not originate until the fourth or fifth week, when the skin has taken on a more definite form. The epidermoid is derived purely from ectodermal, the dermoid from ectodermal and mesodermal elements. The presence of hairs is, therefore, pathognomonic of dermoids.

In the 39 cases cited by Brock and Klenke 33 per cent. occurred in the posterior fossa, in the mid-line. The temporal lobe was the next commonest site (15 per cent.), and only 13 per cent. of all the growths communicated with the lateral ventrical. 28 per cent. of cases arose in the first decade, and 23 per cent. between 20 and 30 years of age; males predominated slightly; most of the growths were single and of pial origin.

The case we have recorded below is therefore typical, and its chief interest appears to us to lie in the extreme length of duration of symptoms and the gradual development of physical and mental changes.

The neoplasm was observed at the autopsy in a youth aged 25, who had been admitted to Southmead Hospital seven days previously comatose and cyanotic.

The patient was born in 1909, and attended Barley Field Elementary School. His mental condition first attracted

attention at 12 years of age, when his case was reported for investigation in regard to his intelligence, with a view to his transfer to a Special School. On account of his age, however, no further steps were taken.

He left school at 14; his mother died a year later; he earned his living selling papers in the street. The money he earned he is said to have spent "in a silly manner." At 18 years of age he was admitted to Stapleton Institution and certified by Dr. Symes under the Mental Deficiency Act as follows: "Face asymmetrical, partial paralysis of right arm and leg. His memory is very poor. He failed on every occasion to repeat seven digits when dictated to him. Writes fairly, reads very imperfectly. Has difficulty in doing any simple calculation or problem in his head. Does not know the difference between a king and a president. He is good at problems of fact. Could not hold his own with other boys of his age on account of his physical and mental infirmities. He is subject to epileptic fits." His intelligence quotient by the Binet Simon scale was about sixty-two.

After his admission his mental condition slowly deteriorated, and he continued to have from fifteen to twenty fits per month, both "minor" and "major" in type. His general physique was good, robust, and well-nourished, but he was liable to acne, boils, and other septic conditions. On 15th March, 1934, he suddenly became exceedingly drowsy, and shortly after developed a squint and ptosis of the left eye. Examination of the discs showed bilateral optic neuritis. On 7th April, 1934, he was transferred to Southmead completely stuporose and somewhat cyanosed. Temperature 96·8°F. Pulse 120 per minute. Respirations 12 to 14 per minute. Examination of the eyes showed a complete ptosis on the left, and almost complete ophthalmoplegia of the left eye. The pupils did not respond to light and were unequal, the left being irregular and larger than the right. The right eye showed nystagmoid movements towards the mid-line. Both discs showed well-marked papillœdema. Pinching of the neck or tapping of the face on the left side resulted in a slow and limited spasm of the left facial muscles. Abdominal reflexes were absent. The right arm showed an old paralytic lesion with contractures; the left arm was freely movable; no tendon reflexes could be elicited either side. The legs showed partial paresis. The reflexes were present and normal. No abnormality was found in the other organs. Attempts at feeding were unsuccessful, save with a tube, and patient died on the 13th April.

Pathology.

At the post-mortem examination, carried out by one of us (P.P.), the right cerebral hemisphere and mid-brain showed no abnormality, and the right lateral ventricle, though distended by cerebro-spinal fluid, contained no foreign material.

The left hemisphere contained a large growth, the situation of which is delineated in the figure. The growth was originally enclosed within a thin membrane, so that actually it was a semi-solid cyst, but the membrane was torn on removing the brain. The temporal lobe was hollowed out on its superior and mesial aspects by the tumour mass, which compressed without invading the brain substance. The hippocampal gyrus and uncus were distinguishable but much distorted. The space formed measured about two and a half by two inches.

The growth consisted of :—

1. A solid, cream-coloured mass of cheesy appearance, fatty consistency, and friable texture. This filled the space above described, but was nowhere adherent to its walls. Scattered throughout its substance were numerous fine, short, silky hairs.

2. Small cauliflower-like masses of growth projecting from the walls of the space, adherent but easily removed.

3. Hairs, growing either singly or in masses ; some were fine, others were coarser and darker.

On opening up the left lateral ventricle this was found to be almost filled with the growth, extending into the anterior, posterior and descending horns. It was mainly of the warty, cauliflower-like type showing no marked evidence of compression. The masses of growth, inside the ventricle and without, connected up through a space at the tip of the inferior horn of the ventricle, apparently formed by a dilation of the lymphoid spaces around the choroid plexus of the lateral ventricle. This space was about 4 mm. in diameter, and was enclosed by a smooth-edged membrane. The brain substance and choroid plexus were free from neoplastic invasion.

The naked-eye appearance and the presence of dermal structures (hairs), together with its relation to the meninges, pointed to the growth being a true dermoid of pial origin—as opposed to the more frequently occurring epidermoid.

PLATE VIII

FIG. 1.
Cerebral Dermoid.

Histology.

Sections taken across the brain tissue at the site where the growth of hair was thickest showed :—

1. Superficially a thin connective tissue layer containing well-formed blood vessels, small round cells, large cells with numerous central nuclei, hairs and hair follicles.

2. Deep to this an almost structureless and œdematous area, containing small round cells and devoid of nerve cells. Newly-formed vessels were present in its substance and one hair was embedded deeply in it.

3. The above area merged into a comparatively normal brain tissue, showing, however, some compression, with increase of neuroglia and round-celled infiltration.

Sections across the mass of growth showed :—

1. Areas of lipoidal material staining well with Scharlach R. and Nile Blue, patches of crystalline cholesterol deposits and myelin droplets.

2. Hairs with normal structure and slightly pigmented core.

3. Finely-fibrillated reticular or neuroglial tissue with associated cells.

4. Nerve cells of the ganglion type, containing granules and surrounded by small monocytes ; also nerve cells similar to those of the cortical cerebral layer.

5. Very many newly-formed blood-vessels, the walls consisting of a single layer of endothelial cells, and the width being that of a red blood cell only.

6. Large cells suggestive of an epidermal or dermal organ, situated mainly in relation to the hair follicles.

Owing to the absence of relatives it has been difficult to obtain adequate details of the patient's past history, and we are greatly indebted to Dr. Datta of Stapleton Institute and to Mr. A. C. J. Gregory of the Bristol Education Department for their help in this matter.

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REFORM OF THE MEDICAL CURRICULUM.

BY

A MEDICAL STUDENT.

FOREWORD.

AT the request of the Editorial Committee some suggestions for applying the Manchester proposals to the methods of clinical teaching at Bristol Medical School have been prepared by a senior student now engaged in clinical work. He has taken the opinions of his fellow-students, and the "suggestions," which are clearly and temperately expressed, may be regarded as representative of the present-day students' views. It should be realized that the medical students, when they reach the "clinical" part of the curriculum, are actually engaged in postgraduate study, except that they have not received a degree for pre-clinical study such as they would have obtained at Oxford or Cambridge.

The Manchester Medical School recently appointed a Clinical Commission to "prepare a report on the clinical instruction of students in Manchester and other medical schools with a view to submitting recommendations thereon to the Medical Board of the Royal Infirmary." This report has been published, and most of its conclusions and recommendations are worthy of consideration by the Medical Boards of many teaching hospitals.

The signatories to the report state that their suggestions are based upon two essential points. "First, that at least 75 per cent. of men engaged in the practice of medicine are general practitioners. Second, that . . . three factors are essential to the successful evolution of a competent general practitioner, namely theoretical knowledge, practical experience, and a sense of responsibility. The basis of training must be a 'learning by doing'; self-reliance and resourcefulness can never be developed in the student by lectures, demonstrations, and other passive educational procedures."

It will be convenient to examine the Manchester proposals in turn, suggesting any possible applications of them to the methods of teaching at present in use in the Bristol Medical School.

The Manchester proposals quoted here are placed in inverted commas.

THE MANCHESTER PROPOSALS.

Lectures.—1. "Attendance at lectures should be voluntary. The system of taking a roll-call at lectures assumes immediately that the lecturer has nothing to offer of sufficient interest to ensure the voluntary attendance of students. . . . If attendance were optional, then the numbers present would be an index of their value in the eyes of the student."

2. "Systematic lectures should be drastically reduced in number. The chief value of systematic lectures is to serve as an introduction to the subject. With this object in view, brief introductory courses of systematic lectures in the elements and principles of the three major subjects might well be introduced."

The present series of Surgery Lectures given at Bristol admirably fulfils this purpose, and after attending them the student feels well equipped to deal with the commoner minor surgical conditions.

3. "Tutorial groups should be established to replace the systematic lecture courses, the groups consisting of as few

students as possible ; their work should invariably be clinical in nature."

4. "Correlation between all the branches of Clinical and Academic work is vitally necessary to prevent overlapping and unnecessary repetition. No attempt is made to correlate the teaching of Pharmacology, Pathology and Bacteriology with the final teaching in Medicine, Surgery and Obstetrics, nor do these three former subjects at any time attempt a specifically clinical approach in their teaching. At present all these subjects of the Final Degree Examination are kept in water-tight compartments, instead of being taught and preserved as the inseparable parts of the whole subject of Medicine, which they really are."

At present there is little or no clinical teaching in Therapeutics at Bristol Medical School, and Pathology is rarely mentioned or discussed in the wards.

In Surgery, and to a lesser extent in Obstetrics as well, there is an admirable series of Museum Classes. These are held once a week in the Museum of the Royal Infirmary : various diseases are discussed in systematic fashion, each condition being illustrated by pathological specimens, and by what is almost as valuable—the personal experiences of the demonstrator.

These classes could be extended. Certain honorary physicians and surgeons specialize in some branch of their work : these "specialists" might give a series of Museum Classes in their particular subject, illustrating them by specimens, personal experience, slides, and, most important of all, by showing cases. Two useful series of classes — one in Genitourinary Surgery, the other in Orthopædics—are given annually at Bristol on the lines suggested above.

A further suggestion is that attendance at these classes should be limited to the clerks or dressers of the demonstrator, and students who are undergoing the final eighteen months of their training.

The systematic lectures in Medicine, Surgery, and Obstetrics might well be replaced by intensive tutorial classes given during the various clerkships and dresserships. These, with the Museum Classes suggested above, would more adequately and satisfactorily cover the ground than do the present series of systematic academic lectures.

Applied Physiology needs more attention at Bristol. Only four or five demonstrations are given annually, and these to students who are about to take their examination in Anatomy and Physiology. A course of clinical demonstrations in this subject might profitably be given to students doing their medical clerkships.

With regard to Anatomy, a revision class in Pure Anatomy, given in the Dissecting Room, would be very useful to students in their final year.

Preventive Medicine is another subject rarely mentioned in the wards. Its practice should receive equal consideration with the treatment of the disease under discussion.

It is probably true to say that students are almost entirely self-taught in the art of interpreting skiagrams. A few demonstrations in this could profitably be included in classes given to clerks and dressers.

Lectures : Conclusions.

1. "That clinical tutorials would be more effective in dealing with any subject which lends itself to this approach than any series of set lectures."

2. "That when lectures must be given, then the function of the lecturer should be : (*a*) To present a view of the subject as a whole ; (*b*) To state principles ; (*c*) To bring text-books up to date ; (*d*) To emphasize salient points in text-books by illustrations from the lecturer's own experience ; (*e*) To vitalize the subject."

"Lectures in Anæsthetics are, from this point of view, quite useless. Lectures in Applied Anatomy and Physiology could be completely replaced by lecture demonstrations and tutorials: the same applies to a large extent to the systematic lectures in Medicine, Surgery and Obstetrics. Acute medical, surgical and gynæcological conditions could be dealt with in a short series of lectures."

THE WARDS.

The arrangements at Bristol, whereby students are allowed to examine patients, are quite satisfactory, although students occasionally find it difficult to obtain the help of nurses when examining female patients.

ARRANGEMENTS FOR STUDENTS IN THE WARDS.

"Students are not entirely unnecessary so far as the running of the hospital is concerned, but they are actually a hindrance to the routine work carried on throughout the hospital."

The first part of this somewhat sweeping statement is certainly true of the Bristol Medical students: the second part is true of the "new student" in the wards or casualty department.

"This is keenly felt by the students and is, we think, the chief factor responsible for the lack of initiative and the general slackness which are so often found among clerks and dressers in the wards."

Most students arriving at the Royal Infirmary or General Hospital find themselves, in the words of the Manchester report:—

"Untutored and apparently unwanted, perpetually in someone else's way, and to all intents and purposes entirely superfluous."

In order to develop in the student practical and theoretical knowledge, with a sense of responsibility, the following scheme for clerkships and dresserships is suggested.

Clerks and dressers should be junior or senior, according to whether it is their first or second three months, respectively, or medical clerking or surgical dressing.

Duties: Medical Clerks, Junior.—Case notes and testing of urines, with instruction by the House Physician, and, later, some practice in the duties they will carry out as seniors.

Seniors.—Supervision of juniors; prescribing of simple drugs and general treatment for own cases, under House Physician's supervision. Obtaining of specimens, and all minor clinical investigations, together with any other general routine treatment.

Surgical Dressers.—An equal or nearly equal number of senior and junior dressers should be attached to each Honorary Surgeon. Each junior to be under guidance and supervision of a senior, and the two to be allotted jointly the usual number of beds.

Junior Dressers.—Case notes and testing of urines, with instruction by House Surgeon and seniors.

Senior Dressers.—Routine treatment of own cases. Pre-operative treatment, certain (*e.g.* perineal, inguinal) dressings, bladder wash-outs, removal of stitches, tubes, etc., obtaining of specimens, writing up of and assisting at operations on own cases. Instruction of juniors in catheterization, venepuncture, bladder irrigation, etc.

The senior clerks and dressers should, so far as possible, be given practice in the prescribing for and treatment of patients.

Theatre.—The senior dressers should be second assistants to their Honorary Surgeons at operations on their own cases, or act as first assistants if the House Surgeon is administering the anæsthetic. They should be responsible for writing up the operation notes. The

junior dressers should take charge of the instruments, leaving the swabs, needles, etc., in charge of the theatre nurse.

At present the Professor of Surgery demonstrates microscope slides of his operation cases once a week at the Infirmary. A useful extension of this would be for the Pathologist to demonstrate slides of all the week's operation cases.

WARD ROUNDS.

These are often so well attended as to defeat their object by debarring many of the students from examining the cases and taking histories. It would be better if ward rounds were made by the Honorary, accompanied only by his houseman, clerks or dressers, and the sister.

WARD CLASSES.

One Ward Class should be held each week in the clinical subjects by each Honorary Surgeon or Physician, attendance being restricted to final year students and the clerks or dressers of the Honorary.

One or two Ward Classes should also be held each week by the Registrar or housemen exclusively for the students working on the unit. The housemen should constantly supervise their students, giving them instruction in diagnosis, treatment, clinical investigation (ward and laboratory), and minor surgery. Further, students should have opportunities of observing and practising routine nursing treatment (enemata, packs, etc.).

It is most important that Ward Classes should not clash.

SPECIAL SUBJECTS.

Ear, Nose and Throat, Eyes, Skin.

The teaching in these subjects is entirely satisfactory. The only complaint one can make

is that the student has no opportunity, except in Dermatological Out-Patients, of carrying out treatment.

Anæsthetics.—At the Bristol Royal Infirmary a student acts as an anæsthetist's clerk for three months, during which time he personally gives as many anæsthetics as possible, the average number being about 100 cases. The institution of a course in Dental Anæsthetics would be beneficial.

Post-mortem Department.—Students should be allowed to assist at post-mortem examinations on their own cases.

Obstetrics.—Teaching in Midwifery and Gynæcology is admirable. The dressers are allowed to perform minor gynæcological operations, such as dilatation and curetage. The Midwifery Clerks, resident for six weeks each in rotation, are permitted to do versions, apply forceps, etc., under the supervision of the Resident Obstetrical Officer. But there is a danger that the Midwifery Department may come more and more to exist for the benefit of nurses taking the C.M.B course, thereby depriving the resident clerks of many cases.

Children's Diseases.—These should be more fully treated by the institution of a series of tutorial classes on the lines indicated for those in other subjects.

Tuberculosis, Infectious Diseases and *Vaccinations* are adequately treated in the Bristol curriculum.

Psychology.—A course in normal and abnormal Psychology should be given, correlated with the course in Mental Diseases.

Orthopædics.

"In Orthopædics the greatest need is for practical tuition in, and experience of, the treatment of fractures, application of splints and plaster, and so forth."

Most of the fracture cases arriving at a Bristol teaching hospital lose their teaching value. The student who sees them in the Casualty Department is usually unversed in the art of treatment, and told by the Casualty Officer to apply such and such a splint. He knows nothing of the mechanical or surgical principles involved, and is usually told nothing of the method of diagnosis and the X-ray appearances: a step in the direction of remedying this defect in the student's training has been the recent establishment of a "fracture clinic" at the Royal Infirmary.

V.D. Clinics.—More attention should be paid to this subject, and students given some opportunity of carrying out treatment.

OUT-PATIENT DEPARTMENT.

"The Out-Patient Department of a teaching hospital is the nearest approach to the General Practitioner's Surgery."

At Bristol Medical School a student attends the Out-Patient Clinic of every Honorary for whom he works. This means that he is present in Out-Patients at least once, usually twice, a week for over two years. In his final year the student is at liberty to attend any clinic he wishes.

At first sight this system would appear to be almost perfect. In practice, however, some disadvantages become apparent. It means that there may be as many as eighteen students present at the Clinic of an Honorary who is known to teach well.

"It is pleasant neither for the patient nor the student to be in a constant state of siege. . . . It is easy, then, to understand that any student who examines more than two cases in a morning is extremely fortunate."

"The control of the attendance of students at these Out-Patients should be exercised by the Clinical Deans. If this suggestion is impracticable . . . students should be

encouraged to visit Out-Patient Departments of other hospitals. . . . It should be made clear that Out-Patients are intended primarily for those in their final year or those who are acting as clerks or dressers to the Physician or Surgeon in charge."

"Finally, all histories of patients should be taken by students. The initial diagnoses, necessary investigations and treatment should be suggested by them."

RESIDENCE OF STUDENTS.

"This is a matter of considerable import . . . a period of residence should be compulsory for students. It is our opinion that the present student, when qualified, may have neither the knowledge nor the experience of these things (medical and surgical emergencies) which a practitioner of medicine should possess before he attempts to enter into the practice of his profession. The purpose of compulsory residence is that the student should acquire this experience and this knowledge."

That Bristol Medical School is ahead of many other schools in the matter of students' residence is seen when comparing the practical knowledge of Bristol-trained medical men with that of men trained at some of the larger teaching hospitals. There is, however, still room for improvement.

The prospectus of the Bristol Medical School states that during their period of residence (one week each, in rotation, during the six months of surgical dressing) students attend to all casualties under the supervision of the house surgeons, and thus have more than ordinary opportunities of learning practical Surgery. But the resident surgical dresser is so pressed by duties —many unnecessary, and some entirely non-clinical— that he has little time to learn any surgery, even if any attempt were made to teach him, during his periods of residence.

The resident dresser is on duty all day and every day during his weeks of residence, when he has

to perform his own clinical work and attend to his own patients in the wards. In addition, he is "on call" for Casualty and for any emergency ward duties. Yet from 9 a.m. to 4 p.m. both the Casualty and non-resident dressers are on duty, and available to attend to the patients allotted to them.

The resident dresser performs, among others, the following duties: Insert stitches, give anæsthetics, incise abscesses, and generally work under the direction of the Casualty Officer, prepare male patients for immediate operations, take all immediate operation cases to the theatre, and he may be allowed to assist at the operation, though this is a privilege now rarely given. After the operation he has to take the case back to a ward. Before midnight he has to take the day's collection of throat swabs, etc., from Casualty to the incubator in the Pathology Laboratory. During the night he is expected to perform catheterizations, bladder irrigations, etc., as may be required.

Without doubt this is excellent training for the arduous post of houseman, but porter's duties might be abolished, or if this is economically impossible, two dressers, senior and junior, might be "resident," with the less responsible duties allotted to the junior. This would enable the resident dresser to pay more attention to the clinical work and derive greater benefit during his period of residence.

SUMMARY.

Briefly summarized, the suggested alterations to the teaching methods and clinical duties at Bristol Medical School are:—

1. The replacement, as far as possible, of systematic lectures by clinical tutorial groups.

2. The correlation of clinical and academic work.

3. The institution of a system of Junior and Senior clerkships and dresserships in Medicine and Surgery.

4. The limitation of attendance at Ward Classes and Out-Patient Clinics to clerks and dressers of the teacher and final year students.

5. The revision of the duties of the resident surgical dressers.

If any alterations in clinical teaching are contemplated at Bristol, the students would greatly appreciate the establishment of a committee, consisting of the Dean of the Faculty and Clinical Deans, together with some student representatives from the Royal Infirmary and General Hospital.

The students are very grateful for this opportunity of expressing their opinions on the subject of reform of the Bristol medical training.

Reviews of Books

A Short History of Ophthalmology. By A. Sorsby, M.D. Pp. vii., 103. Illustrated. London: John Bale, Sons and Danielsson, Ltd. 1933. Price 3s. 6d.—This very interesting little book contains the history of ophthalmology from about 2000 B.C. down to the end of last century. The introductory chapter deals with the dawn of ophthalmology in the ancient East—Babylon and Egypt. According to the Ebers papyrus (*c.* 1550 B.C.) the Egyptians knew such conditions as blepharitis, chalazion, iritis, cataract, and dacryocystitis, but almost the only surgical procedure it mentions is epilation. Hardly anything was known of the anatomy of the eye. The slow development of ophthalmology is traced in Greece, Alexandria and Rome. After the Mohammedan incursions ophthalmology seems to have taken a new lease of life. Hospitals, departmentalized very much in the modern manner, grew up and ophthalmic departments were always large and important. The chapters devoted to the anatomy, physiology and pathology of the eye show that it was not till the time of Leonardo da Vinci and Porta that the eye came to be regarded as a *camera obscura*. Mariotte discovered the "blind spot" in 1668. Porterfield showed, in 1759, that the blind spot was the entry of the optic nerve and that the retina was the essential organ of sight. The chapter on cataract contains a very good coloured illustration of the Indian operation of "couching"—the usual operation till Dairel published his account of the operation for extraction of the lens in 1748. Glaucoma has a chapter devoted to it. No definite conception of its nature existed till about 1840. In the chapter on therapeutics it is recorded that it was Augustin Prichard, of Bristol, who in 1851 resorted to excision of the eye to prevent sympathetic ophthalmia. There are chapters on spectacles, the ophthalmoscope and ophthalmology in the British Isles. The last gives a good deal of information about the notorious quack oculists and charlatans of the seventeenth and eighteenth centuries. There is a bibliography of the history of ophthalmology, and there are indexes of names and subjects.

Surgical Anatomy and Physiology. By N. C. LAKE, M.D., M.S., D.Sc., and C. J. MARSHALL, M.D., M.S. Pp. ix., 888. Illustrated. London: H. K. Lewis & Co. Ltd. 1934. Price 30s.—A text-book of nearly 900 pages is a formidable addition to the medical student's library, and this volume will probably find its place rather as a work of reference than as the daily companion of the embryo surgeon. In this capacity it is truly excellent, although the emphasis is distinctly on anatomy rather than surgery or physiology. The diagrams are clear and well chosen, and include, beside many old friends, a number of skiagrams that are particularly valuable from the clinical side.

Surgical Applied Anatomy. By SIR FREDERICK TREVES, Bart. Ninth edition. Revised by C. C. CHOYCE, M.D. Pp. x., 720. Illustrated. London: Cassell & Co. Ltd. 1934. Price 14s.—The appearance of a ninth edition of a work of this kind just fifty-one years after the first is, in itself, sufficient testimony that the book in question has filled an important place in the student's library for nearly two whole generations. This fact is not to be wondered at, for to many thousands of readers "Treves" has long been recognized as an old friend. The study of Applied Anatomy is not too popular with students, and a readable book on what is really a most important subject is badly needed. This book is essentially readable, and that is one reason why it has retained its popularity for so long a period of time. The author of this edition has departed from what he calls Treves's "anecdotal" style, and although in some ways that is to be regretted, the decision on the whole is probably a wise one. It may be said that the whole work has been thoroughly and competently revised, and that is nearly all that need be said. In its new guise it will continue faithfully to serve the present generation as it has an earlier one. A careful search reveals nothing omitted that might usefully have been included, and only one mistake—a misprint in the fourth line of page 468.

A Synopsis of Medicine. By H. L. TIDY, M.D. Sixth Edition. Pp. xvi., 1,112. Bristol: John Wright & Sons Ltd. 1934. Price 21s.—The sixth edition of Tidy's *Synopsis* is an additional proof that this book is very widely appreciated. The advances made in the four years since the fifth edition was published have made extensive revision necessary. This has been very well carried out. The descriptions given of the disorders resulting from disturbances of the parathyroid

functions deserve the highest praise. So too does the chapter on the anæmias. Additions to our knowledge of the blood and its disorders are being made so rapidly that the difficulties of differentiating and classifying the anæmias are immense, but the author's plan offers a very solid foundation from which to follow future progress. There is no need to enumerate all the new matter. A glance at the preface will satisfy most of our readers of the necessity of getting the new edition.

Industrial Maladies. By SIR THOMAS LEGGE, M.D. Pp. xvi., 234. Illustrated. London: Oxford University Press. 1934. Price 12s. 6d.—Workmen's compensation is very definitely on the increase, and more and more cases are seen by both general practitioners and specialists, which demand not only a knowledge of industrial maladies, but of the processes and agents by which they are brought about. This book deserves, therefore, to be widely read, and will be particularly appreciated by certifying surgeons. The preface contains a life history and an account of the great work of Sir Thomas Legge. The subject-matter is well presented in fifteen chapters, and deals with industrial poisoning by lead, phosphorus, mercury, arsenic, and other metallic compounds. Dermatitis, pulmonary diseases due to dust, and tar cancer are thoroughly considered. There are thirteen good photographic plates illustrating these. The author does not introduce the more surgical conditions, hence there is no account of bursitis, or injuries to the back, and like conditions. The book makes interesting reading, as it contains historical notes on several diseases—a good account of glass blowers' cataract is given. The graphs are disappointing and are difficult to read, and a better method of presenting graph 9 might be found. Not everyone will agree with some of the views expressed, *e.g.* page 51: "There is a fairly close relation between the degree of exposure to lead and the proportion found in the urine and the medical condition of the exposed persons." This has been found on very many occasions not to be true. The index is good, and the book should prove a valuable one for reference. The work of such an eminent authority is worthy of addition to the libraries of all those more directly concerned with industrial diseases.

Diffuse Sclerosis. By L. BOUMAN, M.D. Pp. iv., 160. Illustrated. Bristol: John Wright & Sons Ltd. 1934. Price 15s.—This book summarizes very completely our present knowledge of a condition which under various synonyms, to

wit, Schilder's disease and encephalitis periaxialis diffusa, has attracted a good deal of attention of late. The disease owes its interest to its relationship with other diseases exhibiting demyelinization of the nervous system, notably disseminated sclerosis and subacute combined degeneration. Regarded as a rarity, one wonders how often it passes unrecognized. Most of the literature on the subject has been scattered about in monographs. Professor Bouman's book puts the subject in a very readable form. It is excellently illustrated and contains a very complete bibliography. After a full discussion of the pathological anatomy and pathophysiology he summarizes the whole, indicating the difficulties of, and cardinal points in, diagnosis. He gives a detailed account of a number of personal cases which have been carefully studied throughout their illness to autopsy, together with excellent microphotographs of sections showing the specific lesions. The correlation of lesions and clinical signs is carefully discussed. While the book will primarily appeal to the neurologist, it should also be of considerable interest to the general reader. Without doubt, as a book of reference, it fulfils a very useful rôle.

Clinical Electrocardiography. By W. EVANS, M.D. Pp. x., 49. Illustrated. London: H. K. Lewis & Co. Ltd. 1934. Price 5s.—It is perhaps hardly an exaggeration to state that to many medical students and practitioners the electrocardiogram and its interpretation is shrouded in mystery, and rather savours of "black magic." This is perhaps because the majority of books dealing with the subject take the reader fairly deeply into the physics of the electrical reactions of contracting muscle. In this handbook, however, Dr. Evans, after a short introduction and a description of a "plan" to be followed in investigating a tracing and the main points to be noted, allows the actual electrocardiograms (of which there are sixty-four) to speak for themselves with the help of short explanatory notes. The book concludes with a series of "test" tracings, the key to which is given on the last pages. The electrocardiograms are perfect examples of the various abnormalities which they illustrate. Their technical quality is that which we have learnt to expect from the cardiac department of the London Hospital, *i.e.* beyond criticism. The book is well produced, and should do much to dispel the illusion that the interpretation of electrocardiograms is a mystic rite.

Diseases of Infancy and Childhood. By LEONARD G. PARSONS, M.D., F.R.C.P., and SEYMOUR BARLING, C.M.G., F.R.C.S. 2 vols. Pp. xxxviii., 1,798. Illustrated. London: Oxford University Press. 1933. Price £4 4s.—An apology is due to the editors and publishers of this treatise. Our review has been unduly delayed by the indisposition of the reviewer. Nevertheless this delay has not been entirely a disadvantage. It enables the reviewer to realize that Parsons and Barling has established a singular pre-eminence amongst books on children's diseases. The authors of the various articles have been well chosen and are all abreast of modern work and thought. It is impossible to do justice to the many good points in the book by quotation. A limited number of appreciative observations must suffice. The idea of opening the book with a chapter on heredity is sound as well as novel, and Dr. Cockayne has managed to compress an intricate subject clearly and intelligibly. The chapters on feeding and nutrition are first-rate, though the use of negro children to illustrate rickets was at first puzzling until we realized that the authors of these sections are working in the great school of Johns Hopkins. Maitland-Jones has contributed several sections, but that on infantile atrophy is outstanding. The acute exanthemata are well described, and one point is deserving of notice, as it is not widely recognized, namely that the administration of scarlatinal anti-toxin prevents or limits desquamation after scarlet fever. Ignorance of this fact sometimes leads to the original diagnosis being questioned. The duration of diphtheria immunity after protective inoculation is not clearly enough stated. In the bibliography to the chapter on tuberculosis Calmette's classic work *L'infection bacillaire et la Tuberculose* is not included, although in the text some mention is made of his *B.C.G.* E. Watson-Williams has written excellently on the diseases of the upper respiratory passages and of the ear. The colour illustrations of the appearances of the *membrana tympani* in various diseased conditions are both artistic and instructive. Reginald Miller has contributed the best modern account of rheumatic fever that we have seen. Lobar pneumonia is well described by Professor A. E. Naish, but it is verging on pedantry to intitle the chapter alveolar pneumonia. K. D. Wilkinson is good on heart disease and particularly lucid on congenital defects of the heart. If we have criticisms to make they are few, but the discussion of the recognition of the pre-paralytic stage of acute anterior polio-myelitis is inadequate and the treatment

of the disease is very poor indeed. The aid of a surgeon was needed here to write about the modern treatment. A singular relic is included in the after-treatment of empyema: "bottle-blowing" is recommended. In these days of radioscopy have the "bottle-blowers" ever seen on the screen what happens to the unaffected lung? The two volumes are produced and illustrated with the care and excellence that characterize all the Oxford medical publications.

The Clinical Study and Treatment of Sick Children. By JOHN THOMSON, M.D., LL.D., and LEONARD FINDLAY, M.D., D.Sc. Fifth Edition. Pp. xxxvi., 1,075. Illustrated. Edinburgh: Oliver & Boyd. 1933. Price 30s.—The fifth edition of this standard text-book on pediatrics has been rewritten and enlarged by Dr. Leonard Findlay. Its position as one of the leading text-books on the subject in the English language was established long ago, and Dr. Findlay is to be congratulated on having fully maintained the reputation of his master's book by this revision. It might possibly have been better to have discarded the division of anæmias into primary and secondary, a distinction which nowadays is almost meaningless. The discussion of rheumatic fever is frankly disappointing. Surely in these days the disease is looked upon as a carditis with a tendency to arthritis, or to chorea or the appearance of sub-cutaneous nodules. The relegation of carditis to the third place in the "chief manifestations" seems old-fashioned. The chapter on congenital syphilis is admirably written and illustrated, though amongst the "traces of past syphilitic disease" the characteristic skin scars of the "Great Pox" are not mentioned, save in the form of scarring after phagedænic ulceration, which is uncommon. Infantile paralysis is not very well described as regards the pre-paralytic stage. The "tripod sign of Amos" or its modification as described by Dr. Macnamara are not given the prominence they deserve. Too much reliance is placed on the value of convalescent serum and too little importance is attached to the need for calling in an orthopædic surgeon as soon as the diagnosis of polio-myelitis is made. The chapters on feeding and nutrition are splendid reading, although the retention of Finkelstein's terms "bilanzstörung" and "dekomposition" in their German form was surely unnecessary. Some compact translation is not unattainable. But, take it all in all, "Thomson & Findlay" is a rare good book.

What to do in Cases of Poisoning. By W. MURRELL, Fourteenth Edition. Pp. vi., 208. London: H. K. Lewis & Co. Ltd. 1934. Price 5s.—It can be safely said that any publication which has reached, as this one has, its fourteenth edition, has found a public, and so is already assured of success. This last edition has been revised, and new poisons have been added. The chapter on antimony poisoning from enamel ware glaze should prove especially interesting in view of the controversy that it has recently raised. As those who have this book will know, the poisons with their symptoms and treatment are set out in alphabetical order, and that together with an elaborate index will make it especially useful in an emergency. No general practitioner should be without this in his desk or on his bookshelf.

High Blood Pressure. By J. F. HALLS DALLY, M.D., M.R.C.P. Third Edition. Pp. 281. Illustrated. London: William Heinemann Ltd. Price 15s. This third edition of *High Blood Pressure*, by Dr. Halls Dally, has been very largely rewritten and many new illustrations have been added. The chapters on sphygmomanometric methods and instruments are particularly valuable. Standard arterial pressures at various ages are discussed in the light of recent knowledge, with the result that these standards are now lowered considerably. The fallacious standard that the proper adult systolic pressure is the age *plus* one hundred finds no support. Dr. Halls Dally gives no normal systolic pressure as being above 150 m.m. at any age. Halls Dally on high blood pressure becomes, with this third edition, an absolutely indispensable book for every practitioner of medicine, and for none more than those engaged in medical examination for life assurance.

The Etiology and Treatment of Spasmodic Bronchial Asthma. By H. G. OLIVER, M.D. Pp. vii., 48. Illustrated. London: H. K. Lewis & Co. Ltd. 1934. Price 3s. 6d.—This little volume is devoted to propounding the thesis that spasmodic asthma is due to a mycotic infection, a monilia being suggested as the causal organism. Those who have had experience in the field of mycoses will realize that such a thesis needs the closest criticism, and the most unmistakable experimental demonstration. Dr. Oliver's " proofs " are so far based only on cultural recognition of the monilia in the sputum of asthmatics.

Diseases of the Skin. By S. E. DORE, M.D., and J. L. FRANKLIN, M.D. Pp. xii., 410. Illustrated. London: Cassell & Co. Ltd. 1934. Price 10s. 6d.—This book provides a remarkably complete text-book of diseases of the skin, and will be welcomed by students and practitioners. Chapters on the structure and functions of the skin and the principles of diagnosis are followed by descriptions of the various diseases, the clinical appearance, ætiology, differential diagnosis, prognosis and treatment of each condition being given. A useful feature is the use of small type for the rarer conditions. There are no coloured plates, but forty-six black and white illustrations are included, all of these being photographs of actual cases. The only criticism which one has to make is that in many cases too little space is devoted to the treatment of the more common conditions, and it is hoped that this fault will be remedied in future editions.

Melancholia in Everyday Practice. By E. L. HOPEWELL-ASH, M.D. Pp. vi., 136. Illustrated. London: John Bale, Sons & Danielsson Ltd. 1934. Price 7s. 6d.—This book will have served a useful purpose if it enables the general practitioner to recognize those cases of melancholia in which the danger of suicide is certain. Its perusal may in consequence save the lives of some useful members of society among their patients who have fallen victims to morbid depression. In giving a careful review of various cases of manic-depressive mental disorder and involutional melancholia, the author demonstrates the importance of mild cases of melancholia that are often missed or diagnosed as physical illnesses. The chapter on treatment is good, but the author has not appreciated the need for carefully graduated occupational therapy prescribed by the doctor. The author rightly insists on compulsory restraint for every patient who threatens suicide, who refuses food or is dangerous, but it is much to be regretted that he insists on certification also. As a large proportion are willing to be treated as voluntary patients under the Board of Control, and temporary orders are available for all who have lost volition, only those few who refuse to be treated require certification.

Atlas of the Commoner Skin Diseases. By H. C. G. SEMON, M.D. Pp. 220. 103 coloured plates. Bristol: John Wright & Sons, Ltd. 1934. Price 42s.—This is a book which moves the reviewer to write with real enthusiasm. It presents us

with over 100 most beautiful plates, shown by coloured photography, illustrating all the common skin diseases. The practitioner who keeps this by him is likely to derive the greatest possible help therefrom in the diagnosis of skin cases in his practice, a class of case that, it must be confessed, the majority of doctors find very difficult. There is a brief description of each condition illustrated, mentioning how such cases may be diagnosed and treated. The author, photographer, and publishers must share in the credit for a truly beautiful piece of work.

Head Injuries. By L. BATHE RAWLING, M.B. Pp. viii., 86. Illustrated. London: Oxford University Press. 1934. Price 7s. 6d.—This monograph on head injuries by an acknowledged master on the subject is of extreme value to the practitioner in an age when the motor-car provides a regular supply of suitable subjects. The question of diagnosis is dealt with in a most practical way, and the pathology is well illustrated by means of illustrations of injured skulls. Treatment of each of the conditions described is adequately discussed, and the various pitfalls that may await the practitioner receive due notice. Altogether this is a very valuable contribution to our knowledge of the subject, and will find its place on the shelves of all progressive surgeons.

L'Opération de Bassini. By PROF. A. CATTERINA (Geneva). Pp. 57. Illustrated. Paris: Félix Alcan. 1934.—This is a monograph on Bassini's operation for the radical cure of inguinal hernia. Professor Catterina emphasizes the advantages of the original operation as described by Bassini himself over the usual modern variations of it. He describes in detail the technique observed by his master in the treatment of each of the layers encountered in the operation. Each step is given a separate paragraph and is illustrated by excellent paintings by Dr. Orazio Gaigher, who is himself a medical man. There are three points in which the operation described varies from the usual English text-book description: one is the removal of the cremasteric fascia; secondly, the definition of the free border of Poupart's ligament; and thirdly, the care taken to incise the transversalis fascia. The free border of the Poupart's ligament is the point to which the interrupted sutures in the conjoint tendon are fixed. The author claims that the careful technique that he has described, if it is followed strictly, should give a higher degree of security from recurrence than appears to be the experience at present of those who use

the present modifications of Bassini's operation. At the end of the book a very complete list of complications associated with inguinal hernia and their treatment is given, each complication being treated by a concise annotation.

Physical Signs in Clinical Surgery. Fourth Edition. By HAMILTON BAILEY, F.R.C.S. Pp. xx., 287. Illustrated. Bristol: John Wright & Sons, Ltd. 1933. Price 21s.—The moment the reader opens this book he will appreciate the reasons for its popularity, which has been so great that in seven years no fewer than four editions have appeared. It consists essentially of a series of excellent surgical tutorial lectures, admirably illustrated by numerous and well-chosen pictures, the value of careful and systematic clinical examination being the theme. It is difficult to find words adequately to express the admiration that this work elicits, and the practitioner who does not possess it either has nothing to learn about surgery or has no desire to improve his professional equipment.

Diseases of Women by Ten Teachers. Edited by COMYNS BERKELEY, M.D., M.C. Fifth Edition. Pp. xii., 568. Illustrated. London: Edward Arnold & Co. 1934. Price 18s.—The fifth edition of *Ten Teachers* brings this sound and reliable work up to date. Its plural authorship ensures a balanced opinion on difficult and controversial points, and informs the reader of the current teaching in the various London schools. There is a valuable section on the endocrinology of the female, in which are set out the constantly changing opinions of physiologists and biochemists on this subject. The chapters on ovarian tumours contain some notable additions. Short accounts are also given of the use of diathermy, X-rays, and radium in the treatment of gynæcological conditions, and the new edition can be confidently recommended to students and practitioners alike.

Aids to Obstetrics. By L. WILLIAMS, M.D., M.S. Tenth Edition. Pp. viii., 219. London: Baillière, Tindall & Cox. 1934. Price 3s. 6d.—This little book is by no means a dull tabulation of the principal facts of obstetrics. In a brisk style, and without the use of telegraphic English, it presents the chief points of the subject, explains difficulties, and also gives several very useful practical hints. Used in conjunction with the larger text-books it should prove of great value to the student.

Handbook of Filterable Viruses. By R. W. FAIRBROTHER, M.D. Pp. ix., 193. London: William Heinemann Ltd. 1934. Price 7s. 6d.—The author of this handbook, well known in this country for his work on the viruses of poliomyelitis, is to be congratulated on having written a concise, readable account of the filterable viruses, in which all important work on this intricate subject is discussed and analysed with commendable impartiality. Technical details are omitted, but this omission makes for a clearer exposition of general principles, and there is no work of this size in which the general reader will find the subject so carefully reviewed.

The B.C.G. Vaccine. By K. N. IRVINE, D.M. Pp. ix., 70. London: Oxford University Press. 1934. Price 5s. — Dr. Neville Irvine's book on B.C.G. Vaccine is not entitled to rank as an authoritative criticism of the value of B.C.G., although it provides interesting grounds for reflecting on the superb indifference which British medicine has shown towards Calmette's work. Dr. Irvine has seen the administration of B.C.G. in New York and has interviewed some Hungarian workers. Chapter 3 indicates that he visited the Institut Pasteur in Paris during Professor Calmette's lifetime, but his visits do not seem to have been very informing. He hints at some doubts about the meaning of the instructions issued from the Institut, but his doubts read as if a few minutes' conversation would have cleared them up. The general impression is that Dr. Irvine's book tends to belittle by faint praise the wonderful efforts of the late Professor Calmette to grapple with the so far unsolved problem of how to protect children against the risk of infection with tuberculosis. We are told: "Though the favourable results in no way exonerate those investigators who first gave the vaccine to children, yet it must be admitted that sufficient work has now been done to prove that B.C.G. vaccine is harmless to the normal infant." Again: "A considerable amount of misunderstanding and opposition might have been avoided if Calmette would state that the B.C.G. is a 'virus fixe' when cultured according to his instructions." This is just what Calmette personally told the reviewer, when he was invited by Calmette to go over to Dublin and try to induce the Irish Free State President to make a public trial of his B.C.G. vaccine. Having known Calmette for many years, one cannot praise his great energy and perseverance enough. He fought to save the lives of

children, and the future will give him his right place among the great ones of this world. Calmette was always hurt at the neglect of his work by English doctors. The prevention of tuberculosis seems to be neglected in the country, and what is now needed is an investigation by our Ministry of Health, or some other public body, into the value of B.C.G., and any other means for protecting children against the risks of infection with tuberculosis. The issue of this book by the Oxford Medical Publications may help to stimulate an intelligent interest in B.C.G. amongst medical men in this country, and for that reason it is warmly welcome.

Materia Medica for Nurses. By A. M. CRAWFORD, M.D. Third Edition. Pp. viii., 100. London: H. K. Lewis & Co. Ltd. 1934. Price 3s. 6d.—This useful little book has now reached its third edition. Its general arrangement has not been altered, except for the introduction of two new chapters, one of which deals with the interpretation of prescriptions. This contains some information of no importance to nurses, *e.g. fiat emulsio*, or *haustus*, or *unguentum* being mere directions to the dispenser; whilst the common Latin abbreviations for "four-hourly," "six-hourly" or the initials "s.o.s." are omitted. No patient will come to harm because the nurse does not know the meaning of *cum* or *aqua bulliens*, but she ought to recognize "q.q.h." Why does Professor Crawford follow *in* with a nominative (or is it an accusative plural) *bis in dies?* The other new chapter consists of brief descriptions of commonly used drugs, including some proprietary preparations. These are badly placed, they should not have been separated from the groups to which they belong, *e.g. liquorice* should have been added to the chapter on vegetable purgatives and *nepenthe* to the opium and morphine chapter. This is a point for which perhaps the publishers and not the author are responsible.

Garrod, Batten and Thursfield's Diseases of Children. By H. THURSFIELD, D.M., F.R.C.P., and D. PATERSON, M.D., F.R.C.P. Third Edition. Pp. 1152. Illustrated. London: Edward Arnold & Co. 1934. Price 50s.—This excellent publication comes out in a third edition with a good many changes and additions to the text and in the illustrations. It has been brought thoroughly up to date and offers a sound

and for the most part an eminently readable account of the best modern practice of pædiatrics. It is obviously impossible in a short review to do more than briefly to indicate the outstanding features of a work of 1,200 closely (but clearly) printed pages compiled by thirty-six eminent authors, each of them an acknowledged authority upon the subject treated of by him. But the practitioner and senior student will here find in nearly every case all that he can reasonably desire to know of the ætiology, pathology, symptomatology, and treatment of any medical disease in children which he may encounter. As a matter of fact, the scope of the work is much more ambitious than an attempt to deal merely with medical diseases of children. There are also chapters on orthopædics, on diseases of bone and joints, and on surgical diseases of other organs, such as the genito-urinary system. Sir H. D. Gillies writes on hare lip and cleft palate—indeed, a wide field of surgery is covered. These chapters on surgical affections and operative procedures are, perhaps, somewhat less satisfactory than the more medical side of the work. It is a task of great difficulty to compress orthopædic surgery, even in broad outline, into eight pages, and there seems to be, sometimes, a lack of balance in the treatment allotted to other diseases. For instance, cryptorchidism, which every practitioner encounters so frequently, receives but twelve lines, with never a hint of any treatment, whereas congenital valvular obstruction of the urethra, surely one of the rarer abnormalities, receives thirty, with an illustration. But these are only minor criticisms, and for the vast majority of the articles one can have nothing but enthusiastic appreciation. The chapter on rickets is a masterly little monograph, the chapters on the nervous system, on the kidney, and intestinal tract are especially good; in fact, where there is such a high general standard it is rather an invidious task to single particular articles out for praise. As an instance of the way in which the book is brought up to date, it may be mentioned that there are two descriptions of Von Gierke's disease (hepato nephromegalia glycogenica) with reference to Worcester Drought's review of the literature in 1933. The insulin-glucose treatment in severe diphtheria is also described. There are, of course, as in every work, minor matters where the view-point of the reader will differ from that of the writer, but no one in search of sound progressive views on diseases of children will be likely to go away disappointed after a perusal of these pages. We congratulate editors, contributors, and not least the publishers, upon a worthy achievement.

The Influence of Heredity on Disease. By L. S. PENROSE, M.A., M.D. Pp. vii., 78. Illustrated. London: H. K. Lewis & Co. Ltd. 1934. Price 5s.—The author has succeeded in furnishing a concise description of the modern methods employed in the study of human genetics, and in view of the intricate mass of data which have to be dealt with, it is not surprising that special statistical methods have been evolved, with the result that the student who is unfamiliar with mathematical reasoning must find these scientific advances difficult to understand. An instance of the help we can derive from Mendelian analysis is the appreciation of what result to expect from an attempt to eradicate an hereditable feature by controlling propagation in a condition such as mental deficiency. Prevention of breeding would only result in a partial reduction of the incidence, the reason being that if 1 per cent. of the population exhibit a recessive character 18 per cent. must possess it in heterozygous form, and these latter individuals will perpetuate the condition. The author lays stress upon the importance of the numerical analysis of sibships and of the recognition of the environment as a significant factor. This essay should serve as a useful guide to the eugenist as to the most direct lines along which to conduct his researches. We congratulate the author upon the lucid exposition of so complex a subject.

Editorial Notes

Winford Orthopædic Hospital.

As the result of the voluntary winding-up of Bristol Housing Ltd., formerly known as Bristol Garden Suburb Ltd., an additional ward is to be built and equipped at the Winford Orthopædic Hospital.

The original company, known as the Bristol Garden Suburb Ltd., was formed in 1909, for the purpose of developing an estate of 26½ acres at Shirehampton on garden suburb lines.

Now that the Government and the municipal authorities have taken the matter in hand so energetically, and so many private builders being forthcoming, the reason for the existence of the company no longer obtains, and it has been decided to wind up its affairs, repaying in full the capital invested with interest to date.

During the twenty-five years of its existence the company has accumulated a modest reserve, and this, together with the increase in the value of the company's ground-rents brought about by the cheapness of money, is sufficient to enable the directors (with the consent of the shareholders, most readily and cheerfully given) to offer to build and equip an additional ward at the Winford Orthopædic Hospital, which is so urgently required.

The actual sum offered is £10,000, and it is estimated that this will cover the cost of erecting and equipping

a new ward with 56 beds, thus making a total of 112 beds available. Such an extension ought to enable the authorities to admit children in the early stages of rheumatic heart disease without the delay which the present long waiting list involves. The chief object of maintaining a heart hospital school at Winford is the early admission of children suffering from rheumatic fever whilst the damage to the heart may be hoped not to be permanent. The more doubtful the heart disease the more useful will be the treatment at Winford, since it is almost certain that rheumatic fever affects every heart in a greater or lesser degree.

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Bicentenary of the Royal Infirmary.

THE Committee of the Royal Infirmary announce that the Bicentenary of the Institution will be celebrated in 1937. The reason for choosing this date is that the Infirmary was formally opened for the reception of patients on 13th December, 1737. Although the date of the foundation has been given as 1735, there exists a "Vellum Book" in which the roll of subscribers is inscribed, and no subscription is given a date prior to 1736. This is the "Vellum Book" mentioned by Munro Smith in his history of the Bristol Royal Infirmary as having been lost. Mr. Ellis Smith has, however, been fortunate enough to find it amongst the minute books and archives.

In 1837 the Committee discussed at several meetings how they should celebrate the Centenary. Apparently nothing was done to mark the occasion, because a dispute arose between the Committee and Medical Staff which occupied their minds. But in the "State" or Annual Report for 1837

one subscriber is shown to have remembered the occasion. On December 16th, 1837, Mrs. Martha Daubeny, of Henbury, sent a "Centenary donation" of £100.

That is how the Centenary of the Bristol Royal Infirmary was kept. It is to be hoped that at the Bicentenary Mrs. Martha Daubeny's example will be borne in mind.

Obituary

VINCENT MIDDLETON COATES, M.C., M.D., M.R.C.P.

THE tragic death of Dr. Vincent Coates has deprived the West Country of a well-known and distinguished figure. Although he himself was born in Edinburgh, all his family associations were with the West Country. His grandfather, Dr. C. M. Coates, was a well-known physician in Bath, while his father, Dr. C. Middleton Coates, spent all his professional life in Somerset. Vincent Coates himself had carried on a successful practice as a physician in his grandfather's house in Bath since 1919.

Coates first sprang to fame as a Rugby footballer. Educated at Monkton Combe and Haileybury, he got his colours during his first year and later captained the latter school. In 1907 he went into residence at Gonville and Caius College, Cambridge, and won his Rugger Blue as a freshman. Knee trouble kept him out of the Varsity team the following year, but he played several times in 1909 although still somewhat hampered, and was a tower of strength for the College as left wing three-quarter both in 1909 and 1910. He played also many times for Bath and for Somerset, and in 1913 was awarded his international cap, playing for England in all five matches.

Coates entered the Bristol Medical School in 1912, working at the Royal Infirmary. He qualified in Medicine in 1915 and immediately joined the R.A.M.C. In 1916 he was awarded the Military Cross in France, and subsequently went to Salonika, where he was in charge of a travelling bacteriological unit. After demobilization he worked for a time at the Royal

Infirmary, Bristol, and was appointed Physician to the Bristol Hospital for Women and Children and Clinical Lecturer in Medicine in the University of Bristol. Shortly after settling in Bath he was appointed Physician to the Royal Mineral Water Hospital and Assistant Physician to the Royal United Hospital, and last April became full Physician to the latter institution. He was Consulting Physician to the Trowbridge and Freshford Hospitals.

Coates, who was an M.D. of Cambridge and a member of the College of Physicians, will be remembered as a brilliant physician. His chief interest was in the rheumatic group of diseases: he had contributed many important papers to medical literature on this subject, and with J. Delicati had published a book on rheumatoid arthritis and its treatment. He took a prominent part in all movements for the promotion of the study of rheumatic diseases and their treatment both in this country and abroad, being a member of the committee of the Royal College of Physicians on arthritis. He was Secretary to the section of Diseases of Children at the British Medical Association Meeting in Bath in 1925. His many friends and colleagues will mourn his loss and miss the stimulating contact with his original and energetic mind.

In 1915 Coates married Ethel Bertha Longuet Layton, a Bristol Royal Infirmary sister, to whom our deepest sympathy is extended.

MARY SHORTRIDGE MULES, L.M.S.S.A.

MARY SHORTRIDGE MULES was a student at the London School of Medicine for Women and the Royal Free Hospital before she came down here as a student at the Royal Infirmary. She qualified in the summer of 1932. For a year after her qualification she assisted her aunts at their private asylum in Devonshire. She then held the post of Assistant Resident Medical Officer at the Babies' Hospital in Manchester for six months, and in the middle of September this year she went to be House Surgeon at Stroud General Hospital, where she developed a spreading cellulitis of the right arm on Saturday, the 6th of October, 1934, and died, in spite of treatment, the following Tuesday morning.

Although a very retiring individual, she had many good qualities and was very hard-working and painstaking, and her clinical work was excellent. She was getting on very well in her profession when her fatal illness started.

Meetings of Societies

Bristol Medico-Chirurgical Society

SESSION 1933–34.

THE Annual Meeting of the Society was held in the Physiological Lecture Theatre of the University on Wednesday, 10th October, at 8.30 p.m.

Dr. Elwin Harris, senr., was in the Chair, and 104 members were present.

The minutes of the previous meeting were read and, with one amendment, were confirmed.

The President then resigned his Chair to his successor, Professor A. Rendle Short. A vote of thanks to the retiring President was proposed by Dr. J. A. Nixon and seconded by Dr. Corfield, and carried with acclamation, and Dr. Elwin Harris replied.

The President then gave his inaugural address on the **"Origin of the Human Race."** A vote of thanks to the President for his address was proposed by Mr. T. Carwardine and seconded by Mr. C. Moore, and this was carried with acclamation.

The annual reports of the Hon. Secretary and Hon. Treasurer and of the Editor of the Society's *Journal* were read and adopted, and Mr. H. L. Shepherd and Mr. A. E. Iles were re-elected as Hon. Secretary and Hon. Treasurer respectively.

Mr. Fitzgibbon proposed, and Dr. Phillips seconded, that the nine members elected to the Committee should be :—

Mr. Clifford Moore.	Dr. Vincent Wood.
Mr. Duncan Wood.	Dr. Arthur Fells.
Dr. A. L. Flemming.	Dr. Richard Clarke.
Dr. H. L. Fuller.	Dr. A. L. Taylor.
Dr. P. S. Martin.	

and the following three gentlemen were asked to serve on the University Library Sub-Committee : Professor Rendle Short, Dr. George Parker and Dr. J. O. Symes.

BRISTOL MEDICO-CHIRURGICAL SOCIETY.

INCOME AND EXPENDITURE ACCOUNT for the year ended 30th September, 1934.

INCOME.	£ s. d.	£ s. d.
Members' Subscriptions ..	311 17 0	
Do., outstanding for 1933–1934	31 10 0	
		343 7 0
Interest on Bank Deposits		2 8 11
Interest on £500 4 per cent. Consolidated Stock, less tax		15 5 0
		361 0 11
Balance, being excess of Expenditure over Income for the year, carried to Balance Sheet		30 7 4
		£391 8 3

EXPENDITURE.	£ s. d.	£ s. d.
Grant to *The Bristol Medico-Chirurgical Journal*		319 19
Librarian—Honorarium		15 0
Miscellaneous Expenses—	£ s. d.	
Goodway–Refreshments	16 16 0	
Postages and Sundries	14 3 2	
Printing and Stationery	9 6 9	
Audit Fee	2 2 0	
Lantern and Cinematograph at Meetings ..	4 4 0	
Lambert—Upholstery ..	3 17 6	
University Marshal ..	1 0 0	
Lewis's Medical and Scientific Library—	£ s. d.	
Subscription	4 4 0	
Postages ..	0 15 6	
	4 19 6	
		56 8
		£391 8

BALANCE SHEET as at 30th September, 1934.

LIABILITIES AND CAPITAL.	£ s. d.	£ s. d.
Sundry Creditors		7 6 8
Capital as at 30th Sept., 1933	778 4 2	
Less Deficit on Income and Expenditure Account for the year	30 7 4	
		747 16 10
		£755 3 6

ASSETS.	£ s. d.	£ s. d.
Cash at Bank—		
Current Account	118 10 4	
Deposit Account	166 2 8	
Cash in hands of Treasurer	1 11 6	
		286 4
£500 4 per cent. Consolidated Stock at cost		437 9
Sundry Debtors—		
Members' Subscriptions for 1933–1934 outstanding ..		31 10
		£755 3

Audited and found correct,
H. E. KEELER,
Chartered Accountant
Auditor.

16–18 Clare Street, Bristol 1,
9th October, 1934

Dr. Charles spoke of the gratitude of the Society to Dr. George Parker for his work as Medical Librarian, and proposed that he should be re-elected, and the proposition was carried unanimously.

The second meeting of the session was held on Wednesday, 14th November, at 8.30 p.m., in the Physiological Lecture Theatre of the University.

Professor A. Rendle Short was in the Chair, and fifty-two members were present.

The minutes of the previous meeting were read and confirmed.

Dr. A. L. Taylor spoke of the Bristol Blood Transfusion Service, in the introduction of which he has taken such an active share.

Mr. Hamilton Bailey, F.R.C.S., then read a paper on **"The Diseases of the Testis,"** and this was illustrated by excellent lantern slides. Mr. Duncan Wood proposed and Mr. Chitty seconded a vote of thanks to Mr. Hamilton Bailey, and this was carried with acclamation.

Extract from Editor's Report.

COST OF THE "JOURNAL."

SOME of our readers who look at the Treasurer's accounts for the past year on opposite page may be alarmed that the cost of the *Journal* has exceeded the total of Members' Subscriptions. The expenditure on the *Journal* has been exceptional owing to the publication of a comprehensive Index to the first fifty volumes. This Index cost sixty-seven pounds, but members of the Society will agree that when a journal has lasted for half a century its value as a reference work is greatly enhanced by the issue of a Single Index containing the whole contents of the previous volumes in compact and accessible form.

The Medical Library of the University of Bristol,

WITH WHICH IS INCORPORATED

The Library of the Bristol Medico-Chirurgical Society.

The following donations have been received since the publication of the list in September, 1934.

December, 1934.

J. Michell Clarke Fund (1)	29 volumes.
Professor E. W. Hey Groves (2)	5 ,,
Dr. Iwao Matsuo (3)	1 volume.
Dr. C. Bruce Perry (4)	1 ,,
Royal College of Surgeons of England (5) ..	1 ,,

Unbound periodicals have also been received from Professor E. W. Hey Groves, Dr. C. Bruce Perry and Dr. J. Odery Symes.

THE ONE HUNDRED AND FORTY-NINTH LIST OF BOOKS.

The figures in round brackets refer to the figures after the names of the donors. The books to which no such figures are attached have either been bought from the Library Fund or received through the *Journal*.

Abt, I. A. .. .. *Pediatrics* .. .. [1932]
Azémar, A. .. .. *Le paragangliome surrénal* .. .. .. (4) 1930
Bastos, M. .. .. *La Osteosintesis* .. .. (2) 1932
Bouman, L. .. .. *Diffuse Sclerosis* 1934
Boyd, W. .. .. *Textbook of Pathology* .. .. .. 2nd Ed. 1934
Brailsford, J. F. .. *Radiology of Bones and Joints* .. .. (1) 1934
Campbell, D. .. .. *Handbook of Therapeutics* .. .. 2nd Ed. 1934
Cantarow, A. .. .. *Calcium Metabolism* .. .. .. 2nd Ed. (1) 1933
Catterina, A. .. .. *L'opération de Bassini pour la hernie inguinale* 1934
Chopra, R. N. .. *Indigenous Drugs of India* 1933
Clark, A. J. .. .. *Applied Pharmacology* .. .. .. 5th Ed. 1933
Clark, W. E. Le G. *Early Forerunners of Man* .. .. .. (1) 1934
Colwell, H. A., and Russ, S. *X-ray and Radium Injuries* .. (1) 1934
Dore, S. E., and Franklin, J. L. *Diseases of the Skin* 1934
Findlay, G. M. .. *Recent Advances in Chemotherapy* 1930
Fleming, A., and Petrie, G. F. *Recent Advances in Vaccine Serum Therapy* .. .. (1) 1934
Fraser, J. E. .. .. *Anatomy of the Skeleton* .. .. 3rd Ed. (1) 1933
Furniss, A. .. .. *Ultra-Violet Therapy* .. .. (1) 1931
Gadelius, B. .. .. *Human Mentality in the Light of Psychiatric Experience* .. .. (1) 1933

Gilford, H. .. .. *The Cancer Problem* 1934
Gould, G. M. .. .. *Pocket Pronouncing Medical Dictionary* 10th Ed. 1934
Graham, S., and Morris, N. *Acidosis and Alkalosis* .. .. .. (1) 1933
Gray, H. .. .. .. *Anatomy, Descriptive and Applied* 25th Ed. (1) 1932
Greenwood, M. .. *Epidemiology, Historical and Experimental* (1) 1932
Griffith, J. P. C., and Mitchell, A. G. *Diseases of Infants* .. .. (1) 1933
Grinker, R. R. .. *Neurology* .. .. (1) [1934]
Hammond, T. E. .. *The Constitution and its Reaction in Health* 1934
Hornibrook, E. A. *Stand Up and Slim Down* .. .. 6th Ed. 1934
Inkster, L. .. .. *The Treatment of Functional Nerve Cases by the Method of Neuro-Induction* 1933
Jameson, W. W., and Parkinson, G. S. *A Synopsis of Hygiene* 4th Ed. (1) 1934
Johnston, T. B. .. *Synopsis of Regional Anatomy* 3rd Ed. (1) 1934
Keith, A. .. .. *Human Embryology and Morphology* 5th Ed. (1) 1933
Kirschner, M. .. .. *Operative Surgery. The Abdomen and Rectum* Trans. by Ravdin .. .. (1) [1933]
Legge, T. .. .. *Industrial Maladies* 1934
Leschke, E. .. .. *Clinical Toxicology.* Trans. by C. P. Stewart and O. Dorrer .. .. (1) 1934
Magnus, G. .. .. *Frakturen u. Luxationen* .. .. 2nd Ed. (2) 1933
Marlin, T. .. .. *Manipulative Treatment* 1934
Matsuo, I. .. .. *Biologische Untersuchungen über Farbstoffe* 1 Bd. (3) 1934
Mennell, J. B. .. *Physical Treatment by Movement, Manipulation and Massage* .. .. 3rd Ed. (1) 1934
Miller, A. .. .. *Modern Advances in Diseases of the Throat* .. 1934
Netto, N. R. .. .. *Acçao do tanaceto sobre a mortricidade do tracto digestivo* .. .. (2) [1933]
New York Academy of Medicine. *Maternal Mortality in New York* (1) 1933
Nordlund, U. .. .. *Über die sog. primäre . . . streptokokken-peritonitis* 1933
Norris, G. W., and Landis, H. R. M. *Diseases of the Chest* 5th Ed. (1) 1933
Paul, N. .. .. .. *Cutaneous Neoplasms* .. .. (1) 1933
Payr, E. .. .. .. *Gelenksteifen u. Gelenkplastik I. Teil.* .. (2) 1934
Peters, J. P., and Van Slyke, D. D. *Quantitative Clinical Chemistry* 2 vols. 1931–1932
Reynolds, F. N. .. *The Relief of Pain in Childbirth* .. .. (1) [1934]
Ribeiro, E. B. .. *Aspectos cirurgicos da cascose dos nervos na lepra* .. .. (2) 1934
Robson, J. M. .. *Recent Advances in Sex and Reproductive Physiology* 1934
Rocaz, C. .. .. *Pink Disease.* Trans. I. J. Wood .. .. (1) 1933
Rogers, J. S. .. .. *Physics for Medical Students* 1933
Rose, W., and Carless, A. *Manual of Surgery* [ed.] by C. P. G. Wakeley and J. B. Hunter. 2 vols. 14th. Ed. (1) 1933
Roxburgh, A. C. .. *Common Skin Diseases* .. .. .. 2nd Ed. 1934
Sollmann, T. .. .. *Manual of Pharmacology* .. .. 4th Ed. (1) 1932

Sorsby, A.	*A Short History of Ophthalmology*		1933
Stewart, T. J.	*Notes on Milk*		1934
Ten Teachers	*Diseases of Women*	5th Ed.	1934
Thomas, E. W. C.	*A Synopsis of Hygiene*		1934
Thompson, C. J. S.	*Lord Lister*	(1)	1934
Tidy, H. L.	*A Synopsis of Medicine*	6th Ed.	1934
Tidy, N. M.	*Massage and Remedial Exercises*	2nd Ed.	1934
Vaughan, J. M.	*The Anæmias*		1934
Wakeley, C. P. G.	*Aids to Operative Surgery*	2nd Ed.	1934
Wellcome Research Institution and the Affiliated Laboratories			1934
Williams, L.	*Aids to Obstetrics*	10th Ed.	1934
Willis, R. A.	*The Spread of Tumours in the Human Body*	(1)	1934

TRANSACTIONS, REPORTS, JOURNALS, ETC.

Annual Report of the Chief Inspector of Factories, etc.		1924–onwards
Medical Directory, 1935		1935
Nature of Disease Journal. Vol. III.		1934
Quarterly Cumulative Index Medicus. Vol. 15		1934
Royal College of Surgeons of England. Calendar, 1934	(5)	1934
Surgical Clinics of North America. Vol. XIV., Nos. 4 and 5		1934
Westminster Hospital Reports. Vol. XXII. (1929–1933)		1934

Publications Received

From EDWARD ARNOLD & Co:

Anæsthesia and Analgesia in Labour. By K. L. LLOYD-WILLIAMS, M.D., B.S. Price 5s.

The Treatment of Common Female Ailments. By F. J. MCCANN, M.D. 3rd Ed. Price 12s. 6d.

Diseases of Children. By A. E. GARROD, D.M., F. E. BATTEN, M.D., M.A., and H. A. THURSFIELD, D.M., M.A. 3rd Ed. Edited by H THURSFIELD and D. PATERSON. Price £2 10s. 0d.

From JOHN BALE, SONS & DANIELSSON, LTD.:

Melancholia in Everyday Practice. By E. L. HOPEWELL-ASH, M.D. Price 7s. 6d.

From CASSELL & Co. LTD.:

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The Rheumatic Diseases. By G. D. KERSLEY, M.A., M.D. Price 6s.

High Blood Pressure. By J. F. H. DALLY, M.A., M.D. 3rd Ed. Price 15s.

Radium and Cancer. By H. S. SOUTTAR, D.M., M.Ch. Price 21s.

What of the Child? By A. KEFALAS, M.A., M.B., Ch.B. Price 5s.

From H. K. LEWIS & CO. LTD.:

Principles in the Treatment of Inflammation. By T. E. HAMMOND. Price 10s. 6d.

Materia Medica for Nurses. By A. M. CRAWFORD, M.D. 3rd Ed. Price 3s. 6d.

The Etiology and Treatment of Spasmodic Bronchial Asthma. By H. G. OLIVER, M.D. Price 3s. 6d.

Westminster Hospital Reports. Vol. XXII. 1929-1933. Price 7s. 6d.

From WILHELM MAUDRICH:

Periodic Fertility and Sterility in Woman. By H. KNAUS. Price RM. 15.

From OXFORD UNIVERSITY PRESS:

B.C.G. Vaccine. By K. N. IRVINE, D.M., M.A., B.Ch. Price 5s.

Head Injuries. By L. B. RAWLING, M.B., B.Ch. Price 7s. 6d.

From THE STUDENT CHRISTIAN MOVEMENT PRESS:

Right Marriage. By F. R. BARRY, C. MULLINS, and D. WHITE, M.A., M.D. Price 6d.

From JOHN WRIGHT & SONS LTD.:

British Journal of Surgery. Vol. XXII., No. 86. October, 1934. Price 12s. 6d.

An Atlas of the Common Skin Diseases. By H. C. G. SEMON, M.A., M.D. Price £2 2s. 0d.

Synopsis of Surgical Anatomy. By A. L. MCGREGOR, 2nd Ed. Price 17s. 6d.

The British Journal of Surgery. Vol. XXII., No. 85. July, 1934. Price 12s. 6d.

From WELLCOME FOUNDATION, LTD.:

The Wellcome Research Institution, London. Exhibits at the Chicago Exposition, 1934.

Local Medical Notes

University of Bristol.—Chair of Anatomy: The vacant Chair of Anatomy has been filled by the appointment of Dr. Samuel Ernest Whitnall, F.R.S. (Canada), D.M., formerly Robert Reford Professor of Anatomy in McGill University.

Chair of Medicine: The vacant Chair of Medicine has been filled by the appointment of C. Bruce Perry, M.D., M.R.C.P.

Chair of Obstetrics: The vacant Chair of Obstetrics has been filled by the appointment of H. J. Drew Smythe, M.D., M.S., F.R.C.S.

Professor Geoffrey Hadfield has resigned the Chair of Pathology, on accepting the St. Bartholomew's Hospital Chair of Pathology in the University of London.

EXAMINATION RESULTS.

Students of the University have recently passed the following examinations :—

M.D.—J. C. Batt.

M.B., Ch.B.—*Supplementary First Examination. Pass :* B. C. E. Aldous, Ruth Appleby, Dorothy M. Ayre, Mary Crago, Sara M. Field-Richards, E. M. Grace, C. J. R. Jacob, Rosemary W. Knowles.

M.B., Ch.B.—*Final Examination.* (*Part I.*) *Pass :* Monica L. Hawkins, B. Isserlin (with distinction in Pathology), J. S. W. Little, N. R Matheson (with distinction in Materia Medica, Pharmacy, Pharmacology, Therapeutics, and Forensic Medicine and Toxicology), E. Philipp, L. A. Schnipelsky, E. Want, E. L. Ward.

M.B., Ch.B.—*Final Examination.* (*Part II.*) *Pass :* Marion L. Tryon (*with Second Class Honours*), R. A. Mathews (*with Second Class Honours*), R. D. Bodman, J. N. Heales, F. J. W. Lewis, A. J. Matheson, Mary G. Thomas, H. James.

B.D.S.—*Second Examination. Pass :* F. P. Ewens.

B.D.S.—*Third Examination. Pass : In Dental Mechanics and Dental Metallurgy :* A. N. Brimelow, C. C. B. Plumley, P. R. Quartley. *In Dental Materia Medica :* C. A. Blanden.

L.D.S.—*Supplementary Preliminary Science. Pass :* C. F. Howell, Peggy M. Rooke, G. H. John, R. V. Wood.

L.D.S.—*First Professional Examination. Pass :* L. K. A. S. Oxley, J. B. Inverdale, J. C. F. Rolls, J. G. Windmill.

L.D.S.—*Second Professional Examination. Pass : In Dental Mechanics and Dental Metallurgy :* J. G. Coates, C. L. Read, A. J. Staple. *In Dental Materia Medica :* D. E. Royal. *In Dental Mechanics and Dental Metallurgy :* M. J. Banbury, R. A. Hilton, T. John.

Conjoint Board.—M.R.C.S., L.R.C.P.—*Pass :* R. T. Moore (*Elementary Biology*), Jane Mackintosh (*Physiology*), Margaret E. M. Slater (*Physiology*), E. J. Lace (*Pharmacology and Materia Medica*), T. H. Taylor (*Pathology*).

L.D.S., R.C.S.—*Final Examination.* (*Part II.*) *Pass :* J. H. G. Morris, J. W. E. Snawdon.

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TONAGEN is most palatable, and is readily acceptable to patients.

DIRECTIONS FOR USE.—One to two large teaspoonfuls to be given three times a day, well mixed with hot milk, porridge, tea or cocoa.

Price.—No. 1 size tins, 1/9, 18/- per doz.; No. 2 size tins, 3/-, 30/- per doz.; No. 3 size tins, 5/-, 51/- per doz. Hospital size tins, 12/6 each. Single tins supplied to the Medical Profession at the dozen rate.

MIO-MALT (Ferris).

Is a standardized preparation of tasteless Vitamins with Extract of Malt. It contains 2,000 units of Vitamin A and 1,000 units of Vitamin D per ounce, in combination with the finest Extract of Malt specially rich in Vitamin B, Albuminoids, Maltose, Natural Phosphates and Diastase.

MIO-MALT is pleasant to take. It does not produce diarrhœa or other digestive troubles, and it may be given to the most delicate children and invalids; it is a perfect substitute in all cases where Cod Liver Oil and its preparations cannot be tolerated, and it may be taken even in large doses for long periods without any ill effects.

MIO-MALT is a valuable tonic food, both for infants and adults, in all cases of debility produced by lack of Vitamins, and it is specially indicated for use during the Winter months to compensate for the lack of sunshine, by building up the system and strengthening the bodily resistance to disease.

DOSE.—One teaspoonful to one tablespoonful according to age two or three times daily after food.

Price.—1 lb. glass jars 1/9 each, 18/- per dozen; 2 lb. glass jars 3/- each, 30/- per dozen; 4 lb. glass jars 5/6 each, 54/- per dozen; 7 lb. glass jars 8/- each, 78/- per dozen.

Single jars supplied to the Medical Profession at the dozen rate.

FERRIS & COMPANY Ltd.

Union St., BRISTOL

Wholesale and Export Druggists and Manufacturing Chemists.